ENVIRONMENTAL TOBACCO SMOKE:

A REVIEW OF THE LITERATURE

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I. INTRODUCTION

The claim that exposure to environmental tobacco smoke (ETS) causes disease in nonsmokers has generated a great deal of public concern. Although a review of the scientific literature indicates that this claim has not been proved, it is used by antismokers in their efforts to make smoking socially unacceptable and, thereby, to create a "smoke-free" society. The importance of the ETS/health issue to the antismoking movement is demonstrated by the comment of the then director of the U.S. Office of Smoking and Health that "of all the issues, this [ETS] is the one that will propel the United States toward a smoke-free society."[1]

The campaign for a "smoke-free" society has been characterized by one sociologist as follows:

> The strategy, quite overtly, is to progressively stigmatize smoking, segregating the smoker in all public places, and eventually to eliminate smoking as a socially acceptable custom. How is this to be made politically palatable? The answer is clear: by suggesting that smoking harms not only the smoker, but various categories of "innocent bystanders."[2]

The questionable and often contradictory nature of studies concerning ETS exposure and disease is largely ignored by the antismoker; the goal is to achieve publicity and arouse emotion.

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In addition, the issue of nonsmoker exposure to ETS has become the means by which antismokers seek to enact legislation regulating smoking in the workplace and other public places. Here again, the strategy of using the ETS/health argument to modify smoking behavior is apparent. For example, the chairperson of the U.S. National Academy of Sciences committee which authored a 1986 report on ETS remarked that "legislation could also be justified, not because it will have much effect on the occurrence of lung cancer among nonsmokers, but because it may motivate some people either to stop smoking or never to start."[3] In 1990, antismoking activists depicted the ETS issue as a "Trojan horse" because, they contended, smokers will benefit "from the significant reductions in smoking frequency that occur with the proliferation of smoking restrictions introduced in the name of concern for the health of nonsmokers."[4]

Indeed, one reviewer has observed that there are even instances in the scientific literature in which calls are made to establish ETS as harmful "as a means to limit smoking."[5] Scientists with personal motivations against smoking now publicly declare that the "lack of definite scientific evidence" [regarding ETS] should not obstruct efforts in "encouraging adults to give up smoking,"[6] and that "the totality of evidence [on ETS and disease in nonsmokers] is sufficient for public health purposes."[7]

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A German professor of social medicine, Dr. J. von Troschke, recently discussed the tendency of some scientists and physicians to "dramatize the discussion about passive smoking."[8] Dr. von Troschke suggested that such behavior may be an attempt "to compensate for the frustrations" encountered in convincing smokers to quit. He observed that "it is natural to convert one's own frustrations into aggression against the resisting smokers and gladly employ every means by which one can bring pressure to bear." One of those means, Dr. von Troschke said, is "the argument on damaging the 'innocent nonsmokers' health.'"

Similarly, Dr. Gio Gori of the Health Policy Center in the U.S. commented:

> The whole ETS question has been raised as a tool in the larger battle against smoking, and therefore, it has been used with the principle that the end justifies the means, and I think as a scientist myself that many people . . . would be rather taken aback by this misuse of science. . . [T]here are many other examples in our society today where science is misused for political reasons, in regulation as well as in establishing public health policy. The justification of course, is usually that we have to take action on the basis of imperfect knowledge for prudent reasons, but I think that many in our society question how far can we go with prudence because prudence itself could become very dangerous beyond a certain point.[9]

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The tactic of using the ETS/health argument to modify smoking behavior also has been criticized in the international press. One columnist for The (London) Times noted that while she agreed in principle with the antismoking movement, she could not condone "the lying that accompanies it." She explained: "The lie is the claim that the health hazards of second-hand smoke have been scientifically established."[10] Similarly, a writer for a German publication called the use of the ETS/health argument an "unethical" tactic intended to instill "anxiety in the nonsmoker" and "feelings of guilt in the smoker."[11]

Certainly, ignorance of scientific procedures and the inability or unwillingness to recognize faulty research methodology have contributed to the controversy on ETS. As one reviewer of the literature wrote in 1987:

> A pluralistic society such as ours has its lobbies. And it is they who have removed the subject of passive smoking from the scientific to the political stage. It would be wrong to impute this to bad intentions. Inadequate or biassed occupation with the problem, or general insecurity with regard to scientific comprehension, are quite enough.[12]

Given the importance of the ETS issue for the antismoking movement, the purpose of this paper is to review the current scientific literature regarding environmental tobacco smoke and disease in nonsmokers. It is intended to identify sources of

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information and misinformation about ETS in an effort to demonstrate that the weight of the available scientific evidence does not justify the claim that ETS causes disease in nonsmokers.

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II. ENVIRONMENTAL TOBACCO SMOKE AND HEALTH CLAIMS

An Overview

Antismokers typically refer to several highly publicized studies as "proof" that tobacco smoke is associated with disease in nonsmokers. The studies most frequently cited are those by Hirayama[1] and Trichopoulos, et al.,[2] who reported that nonsmokers married to smokers have a higher risk of developing lung cancer, and by White and Froeb,[3] who asserted that nonsmokers with long-term exposure to tobacco smoke in the workplace have significantly reduced small-airways function. Other reports have suggested that children and individuals with lung and heart disease are adversely affected by exposure to environmental tobacco smoke.

In addition, government health officials have issued reports which antismokers are using to pressure legislative bodies in the promotion of a "smoke-free" society. In 1986, two such reports in the U.S., one issued by a National Academy of Sciences committee to the U.S. Environmental Protection Agency (the NAS Report),[4] and the other by the Office of the Surgeon General,[5] concluded that ETS causes disease in nonsmokers. In 1988, the Independent Scientific Committee on Smoking and Health in the United Kingdom issued its report with conclusions similar to those of the U.S. Surgeon General.[6] Most recently, the U.S. Environmental

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Protection Agency (EPA) issued for public review a draft risk assessment on ETS in which it recommended that ETS should be classified as a "known human carcinogen," that is, a cancer-causing substance.[7]

However, critical analyses of these studies and reports by physicians and scientists from around the world, together with other studies which report no association between ETS and human disease, indicate that such extreme health claims are highly suspect and not justified on a scientific basis. For example, in October, 1986, an international gathering of scientists in Essen, Germany, considered the experimental and toxicological findings regarding ETS.[8] Professor J.G. Gostomzyk, director of the Health Bureau in Augsburg, Germany, concluded in his review of the proceedings that "so far, even toxicology has not been able to ascertain with any greater degree of probability than did epidemiology that there exists a link between damage to health and passive smoking."[9]

In May of 1988, organizers of a symposium on ETS in Austria, entitled "Illness Due to Passive Smoking?," issued a press release in which they concluded that "a causal relationship between ETS and illness cannot be established" and that "there is no positive evidence that cancer and other such illnesses are caused by passive smoking."[10]

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In his introduction to the published proceedings of a symposium on indoor air quality held in Argentina in 1988, Dr. Osvaldo Fustinoni, vice-president of the National Academy of Sciences in Buenos Aires, wrote that because of the uncertainty regarding scientific data on ETS exposures and health effects, "it is in question to what extent governments should establish regulations affecting individual freedom, legislating over private facilities like factories or cinemas, theatres, etc."[11]

In his "Summary and Concluding Remarks" from the proceedings of the 1989 International Symposium on ETS held at McGill University in Montreal, Canada, co-organizer Dr. Joseph Wu concluded that the published data on ETS, when critically examined and evaluated, do not provide a scientific justification for the claim that ETS is a cause of disease in nonsmokers.[12]

Dr. Hitoshi Kasuga, organizer of The International Conference on Indoor Air Quality in Tokyo, Japan, concluded in the proceedings published in 1990:

> Most participants were of the opinion that it would be very difficult at the present time to reasonably establish a correlation between passive smoking and lung cancer because a detailed examination of all published data (statistical bias in relationship between ETS and lung cancer, estimated ETS levels, incidence of lung cancer in nonsmokers, histological types, etc.) shows that the relationship, if one exists at all, is very slight.[13]

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Lung Cancer

Although 29 studies on ETS and lung cancer with varying conclusions have been published to date, two highly publicized articles published in 1981 are still among the most frequently cited to support the claim that ETS exposure increases the nonsmoker's risk of lung cancer. A study of Japanese women by Dr. Takeshi Hirayama reported that nonsmoking wives of heavy smokers had a much greater risk of developing lung cancer than nonsmoking wives of nonsmokers.[1] In a study of Greek women, Trichopoulos, et al., concluded that a nonsmoking woman whose husband smoked had twice the risk of developing lung cancer as a nonsmoking woman married to a nonsmoker.[2]

Both studies have been widely criticized in the scientific literature. Numerous inadequacies and inconsistencies in the Hirayama study have been pointed out by noted scientists and physicians who have questioned the validity of its conclusions.[3-25] Dr. Ragnar Rylander, the organizer of the 1983 University of Geneva symposium on ETS, noted that the study had been criticized for its lack of questionnaire reliability, absence of histological or microscopic diagnosis, questionable statistical treatment, and failure to examine such factors as air pollution from heating and cooking.[24] A participant at the International Conference on Indoor Air Quality held in Tokyo in 1987 observed that Hirayama's study

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had been questioned for bias in statistical processing, as well as for failures to measure ETS exposure levels and to evaluate and report "dietary and occupational factors known to influence susceptibility to lung cancer."[6]

Moreover, several researchers have criticized Hirayama's techniques of age adjustment, an important consideration since age may act as a confounding or biasing factor in such calculations.[4-5,11-13] For instance, two studies presented at the Conference on Indoor and Ambient Air Quality, held at London's Imperial College in 1988, reported that Hirayama had not divided his study population into appropriate age groups.[4,11] Elsewhere, the authors of one of these studies noted that the risk Hirayama had reported for nonsmoking wives of smokers disappeared when such age bias was removed.[5] In a 1989 paper, another scientist also demonstrated the effect of age bias as well as a significant interaction between age and husband's smoking status in Hirayama's data.[25] At a conference held in Brussels in 1989, biostatistician Dr. S. James Kilpatrick reported that Hirayama would not have calculated a statistically significant elevated risk if he had used an appropriate statistical model and had adjusted his data by the wife's age rather than the husband's.[12] Hirayama has failed to address these and other issues raised by his critics, and, in fact, published a monograph in 1990 which again contains the criticized analyses and tables.[26]

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Criticisms of the Trichopoulos, et al., study were acknowledged by the authors themselves in an update of their study, published two years after the original report.[27] In that update, they noted that their research had been "criticized (by ourselves and others) because of the small number of subjects, because several tumors lacked histological confirmation, and because controls and cases were from different hospitals." Two German scientists went so far as to describe the study as a "textbook example of how a case-control study should not be performed."[28] Nevertheless, like Hirayama, the Trichopoulos group published another paper in 1990, which appears to use methods nearly identical to those used in the first study; perhaps not surprisingly, it also reports a statistically significant increase* in lung cancer risk associated with marriage to a smoker.[29]

Questions about the reported findings of Hirayama and Trichopoulos, et al., were also prompted by their inconsistency with the conclusions of another study published that same year.

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* A test for statistical significance permits an investigator to say that the results obtained in an investigation, in all probability, did not occur by chance. A statistically significant result also permits the investigator to say that the data in the study support rejection of the hypothesis that there is no association between ETS exposure and lung cancer in nonsmokers. A result which is not statistically significant, however, is consistent with the hypothesis that there is no effect. The data in such a study do not support rejection of the hypothesis that there is no association.

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In late 1981, Lawrence Garfinkel, an official with the American Cancer Society (ACS), reported the results from a follow-up of the group's long-term lifestyle study, involving nearly 180,000 nonsmoking American women divided into categories based on the amount their husbands smoked.[30] By comparing the lung cancer mortality rates of women reportedly exposed to different levels of tobacco smoke, he determined that none of the differences observed were statistically significant and that "compared to nonsmoking women married to nonsmoking husbands, nonsmokers married to smoking husbands showed very little, if any, increased risk of lung cancer." Moreover, Garfinkel cautioned that any study which classified the wives' exposure to ETS solely upon the basis of their husbands' smoking habits could not account for the wives' total exposure and that, consequently, the results could be misleading. (For a discussion of somewhat different data from Garfinkel, see Garfinkel, et al., 1985, at p. 17 of this document.)

Although several subsequent studies have claimed to substantiate the reported findings of Hirayama and Trichopoulos, et al.,[31-36,38-39] a close examination of the methods and conclusions of these studies reveals that their claims are scientifically unfounded. For example, a report by Knoth, et al., which identified 39 nonsmoker lung cancer cases, asserted that exposure to ETS was the "most plausible explanation" for the reported lung cancers.[36] However, the study failed to provide a comparison group as a

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control population. One reviewer consequently characterized the report as containing "only tentative conclusions based on poor data analyzed by unacceptable methods."[37]

In a 1983 study, Correa, et al., reported finding an increased risk of lung cancer for nonsmokers married to smokers in the state of Louisiana in the southern United States.[38] However, the results were based on a small number of cases and did not take into account occupational and indoor and outdoor exposures or any other confounding variables. Furthermore, one of the co-authors of this study later issued reports with contradictory findings for a similar population in a neighboring state.[40-41]

The complex nature of the scientific data in this area is further illustrated by the mixed results of some studies; that is, certain data in a study may suggest a relationship between ETS and lung cancer while other data from the same study may not. For example, in a report on the preliminary results of a health survey conducted in two urban communities in Scotland, Gillis, et al., noted that of the six lung cancer deaths observed among male nonsmokers, four were in those who had reported exposure to ETS.[42] However, no dose-response relationship was noted for females in the study. In a follow-up study published in 1989, the authors provided additional data on lung cancer, as well as a number of other diseases and symptoms.[43] Although the additional data

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suggested a positive association between exposure to ETS and lung cancer in nonsmokers, the results were not statistically significant.

Kabat and Wynder, in a 1984 study in the United States of 25 male and 53 female nonsmoker lung cancer cases, reported a greater amount of ETS exposure at work for male cases compared to the controls.[44] However, no significant effect was reported for males exposed to ETS at home or for female cases exposed either at work or at home. Their conclusion for males was of marginal statistical significance; it is the only statistically significant result among the 11 studies in the published literature which have investigated workplace exposure.

In 1985, Garfinkel, et al., published a study of 134 nonsmoking women with lung cancer who were selected from hospital records during the period 1971 to 1981.[45] The paper provided two analyses of the same data which produced apparently contradictory results. While a statistically significant dose-response relationship was reported between nonsmoking female lung cancer patients and the number of cigarettes smoked per day by their spouses, no significant relationship was reported for the occurrence of lung cancer in nonsmoking women and the total number of hours per day of exposure to ETS over either the past five or 25 years.

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In 1988, Shimizu, et al., reported elevated risks for lung cancer among nonsmoking Japanese women if either their mothers or fathers-in-law smoked, but "no association was observed" among nonsmoking wives of smokers who reported ETS exposures either at home or at work.[46]

In a 1988 case-control study of Chinese women, Geng, et al., reported a statistically significant association between lung cancer in nonsmoking women and spousal smoking. However, no significant associations were reported between lung cancer and exposure to ETS from parents or colleagues.[47]

Inoue and Hirayama reported the results of a case-control study on lung cancer in nonsmoking women from two cities in Japan.[48] The authors reported a marginally significant increased lung cancer risk among nonsmoking women whose husbands smoked more than 20 cigarettes per day. However, the data revealed no significant associations for lung cancer in relation to spousal smoking of less than 19 cigarettes per day, or for the overall category of "being married to a smoker."

In one of the most highly publicized articles in this area, U.S. researcher Dwight Janerich and his colleagues reported that household exposure of 25 or more "smoker years" during

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childhood and adolescence could double the risk of lung cancer for adult nonsmokers.[49] However, the study also noted that exposures to spousal smoking, smoking in the workplace, or smoking in social settings were not significantly associated with nonsmoker lung cancer incidence, nor were cumulative exposures during adulthood or over a lifetime. In a letter criticizing the study, British statistician and epidemiologist Peter Lee wrote: "To base conclusions on the single index that shows a marginally significant positive relation is a misrepresentation of findings from what is an essentially null study."[50] The study was also criticized for its failure to account fully for other confounding factors.[51-52] Additionally, the first report on this study, an unpublished doctoral or Ph.D. dissertation, did not even consider ETS exposure in childhood separately; it reported a statistically significant risk estimate only for 175 or more person/years of exposure.[53] (This number was calculated by multiplying the number of smokers in the household by the number of years nonsmokers spent living with them.) The Janerich, et al., study is the only one of nine published studies investigating childhood ETS exposure to report a statistically significant increase in risk.

Still other studies have reported no significant association between ETS and lung cancer. These include Chan and Fung's 1982 study from Hong Kong which reported fewer "passive smokers" among lung cancer patients than among controls.[54] The

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authors noted that "this finding is at variance with that of Dr. Hirayama's."

Similarly, Koo, et al., studied 200 female lung cancer cases among Chinese women and concluded that ETS exposure in the home or at work had no statistically significant impact on lung cancer incidence.[55] In a second study published three years later, this research group calculated total ETS exposures at home and at work for their population of nonsmoking female patients. They observed no significant disease trends for any of the lifetime measurements of exposure to ETS.[56]

In 1985, Wu, et al., using data from a study of female lung cancer cases in a large U.S. metropolitan area, reported that the lung cancer risk among the 31 nonsmokers in the study population was not affected by ETS exposure.[57]

Lloyd, et al., published results of a study undertaken to investigate the occurrence of lung cancer in an industrial area in Scotland.[58] The authors reported that ETS exposures were not significantly different between the cases and controls in the study; in other words, they reportedly found that ETS exposures appeared to play no role in the increased incidence of lung cancer in the study population.

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Also in 1986, Lee and colleagues investigated the potential role of ETS on the incidence of nonsmoker lung cancer in England.[59] The researchers observed no significant trends in increased risk for lung cancer patients who reported exposure to ETS. In fact, in some disease categories, more controls than cases reported exposure to ETS.

Preliminary data from Geoffrey Kabat's on-going American Health Foundation study of 90 nonsmoker lung cancer cases, presented at a 1990 scientific meeting, indicate no statistically significant associations for lung cancer among males or females who were exposed to ETS during childhood or adulthood (in the home or the workplace).[60]

Sobue and colleagues, in a 1990 study of lung cancer among nonsmoking women in Osaka, Japan, reported no statistically significant associations between ETS exposures during childhood or adulthood and lung cancer among nonsmokers.[61] However, the researchers did note a statistically significant increased risk of lung cancer among women who had used wood or straw as cooking fuels.

In 1989, Swedish researchers Svensson, et al., assessed the relationship between ETS exposure and lung cancer among 38 never-smoking women.[62] They reported no statistically significant

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associations between lung cancer incidence and ETS exposures in childhood, in adulthood or over a lifetime.

The Wu-Williams, et al., study, published in 1990, is the largest Asian case-control study to date; it was conducted in two cities in northeast China by U.S. and Chinese researchers.[63] A statistically significant negative association was reported for spousal smoking and lung cancer in a sample of more than 200 lung cancer cases, suggesting that those women reporting exposures to ETS actually had a lower risk of lung cancer than those not exposed. Other lifestyle variables, e.g., use of coal-fired heating devices, traditional Chinese cooking practices, medical histories, and occupational exposures, were reported to be positively associated with lung cancer incidence.

Another study, conducted by Liu, He and Chapman in Xuanwei, China, reported no increase in lung cancer risk associated with ETS exposure, measured as the presence of a smoker in the household. A number of other factors, however, were reported to be associated with an increased risk for nonsmokers, including chronic bronchitis, family history of lung cancer and cooking over coal-burning stoves.[64-65]

In addition, relatively few studies have considered ETS exposures outside the home; only four studies sought information

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about total exposure to ETS from various sources.[45,55-56,59] Interestingly, none of these studies reported finding a significant association between total ETS exposure and lung cancer.

Meta-Analysis of ETS Studies

Perhaps as a result of the failure of most individual studies to report consistently significant results, certain researchers have recently begun using another method of statistical analysis which combines the reported results from numerous studies. With this method, called "meta-analysis," they claim to have calculated estimated excess risks for nonsmokers exposed to ETS that are 10 to 50 percent greater than those for nonexposed nonsmokers. In basic terms, they have reached these broad conclusions or "generalizations" by calculating an average of the relative risks for nonsmoker lung cancer cases reported by those studies.[66-70]

However, this method of generalization has been criticized for its questionable application to the epidemiological studies on ETS, due to their wide variety of study designs, population selections, techniques of analysis and results.[71-73] It also ignores basic methodologic weaknesses which, according to one governmental report, are characteristic of all the published studies on ETS and lung cancer.[74]

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This method further ignores the fact that the majority of the studies considered in the various meta-analyses do not report statistically significant increases in lung cancer risks for nonsmokers. For example, only two of the 13 studies considered by the 1986 NAS Report achieved statistical significance.[75] (Although the authors of several studies report risks which are not statistically significant, they report "positive" trends in their data. However, trends which fail the test for statistical significance cannot properly be characterized as disproving the null hypothesis or the hypothesis that there is no relationship between ETS exposures and lung cancer). Indeed, of the 29 studies on ETS and lung cancer in nonsmokers published to date, only six report statistically significant risk estimates for the exposure index of spousal smoking. In addition to considering flaws in the individual studies, it has been pointed out that meta-analyses themselves "should combine the careful thought and synthesis of a good review with the scientific rigor of a good experiment."[76]

The epidemiological studies on ETS cited in these meta-analyses were recently assessed by Letzel and coworkers for their scientific rigor; they also performed their own meta-analysis on the data pooled from those studies.[73] Of the 1,023 possible combinations of the 10 case-control studies they examined, only 24 gave rise to statistically significant results. Moreover, the

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statistical significance in those 24 combinations was dominated by the presence of three studies which have been described as of questionable scientific merit.[77] Letzel, et al., characterized the quality of the individual studies as "highly variable and sometimes poor."[28,73] They concluded that any computed excess risk would be negligible and, therefore, could not be used to support the claim that ETS exposures increase the risk of lung cancer in nonsmokers.

A critical review of meta-analysis as an analytic technique was published in 1990 by U.S. medical school researchers Fleiss and Gross.[78] In their review, they detailed the criteria for conducting an appropriate meta-analysis, and demonstrated that, when only U.S. studies of ETS and lung cancer were combined for reasons, among others, of homogeneity or similarity, a statistically nonsignificant summary risk estimate was calculated. Consequently, they concluded that "given the biases that exist in each individual study, the safest conclusion from the present meta-analysis is a negative one: There is no convincing scientific evidence from the epidemiological literature of an association between exposure to ETS and the risk of lung cancer in the U.S."

A similar conclusion was reached by U.S. statisticians Maxwell Layard and Maurice LeVois in their follow-up comments to the draft risk assessment on ETS and lung cancer issued by the U.S.

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Environmental Protection Agency (EPA) in 1990.[79-80] The authors of the draft report performed a meta-analysis of 23 studies which resulted in a significant risk estimate for nonsmokers exposed to ETS. However, Layard and LeVois conducted an expanded meta-analysis, utilizing the EPA's methodology, that included three new studies which had appeared after the EPA staff prepared its document.[79] They calculated a summary relative risk which was "not statistically significant" and asserted that "the addition of the newly available epidemiology makes untenable the conclusion reached in the draft report concerning the ETS/nonsmoker lung cancer epidemiology."

Bias and Confounding Factors

The studies on ETS and lung cancer also have been criticized for their failure to account for a variety of biases and confounding factors that could affect the validity of their reported findings. In their discussion of such studies, for example, American researchers Ernst Wynder and Geoffrey Kabat cautioned that "if the observed relative risk is small," which they note is "the case" in the relationship between ETS and lung cancer, "it is important to determine whether the effect could be due to biased selection of subjects, confounding, biased reporting, or anomalies of particular subgroups."[81]

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Statistician and epidemiologist Peter Lee has argued that the increased risks reported in the various meta-analyses are the result of an inherent bias in study design rather than the result of any genuine effect from exposure to ETS.[82-89] In his analyses, Lee has presented data which indicate that the reported risks cannot be explained on the basis of either ETS exposure or dose for the nonsmoker. Rather, he contends that the reported "risks" are the result of bias caused by a small number of smokers who are misclassified in the studies as nonsmokers.

Other kinds of misclassification may contribute to the reported increase in lung cancer risks among nonsmokers, according to several scientists. For example, none of the studies on ETS and lung cancer provides direct observational information on ETS exposures. Instead, spouses, next-of-kin or friends were asked to estimate the amount of ETS to which they thought the subject was exposed. Such estimates may result in what is called exposure misclassification,[90] which Garfinkel,[45] Friedman[91] and others[92-95] have reported leads to improper indices of exposure and incorrect estimations of risk. In Garfinkel's study, for example, the reported risk for lung cancer in the women exposed to ETS was actually less than for women not exposed when either the women's or their husbands' estimates were used.[45] Other authors, including the National Research Council, have recently criticized questionnaires used in ETS studies for not being standardized or

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validated; they pointed out that misclassification of exposure may occur if the questionnaire is not appropriately designed.[96-98]

Biostatistician Kilpatrick has identified another form of misclassification, called differential misclassification, which results "from the tendency of respondents to inflate the amount of ETS exposure for lung cancer cases and deflate the report of exposure for controls."[90] Similarly, Dr. Ernst Wynder, President of the American Health Foundation, notes that "relatives of a nonsmoking lung cancer patient are more likely to report passive inhalation exposure on the part of their relative than are relatives of a control patient."[99] Wynder and a co-author also noted that even if the patient herself is interviewed, some overreporting of ETS exposure may occur.[100]

Misclassification of case status can also arise if diagnoses of lung cancer are not histologically confirmed.[101] Without histologic confirmation, a tumor arising at another site and metastasizing or traveling to the lung could be erroneously identified as a primary lung tumor.

A more subtle form of potential bias, known as "publication bias," stems from the apparent unwillingness of scientific and medical journals to publish studies which report negative or weakly positive results.[71,102-103] Scientists have

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recently expressed concern over the growing trend among such journals to overemphasize (and hence to publish) only those studies which report positive increases in risk.[104-105] Published studies which are combined for meta-analyses, therefore, may not truly represent all investigations on the issue of ETS exposure and lung cancer.[106]

Moreover, most of the epidemiological studies on ETS and lung cancer have failed to consider age differences, diet, occupation and exposures to indoor or outdoor pollution as potential confounding elements. The importance of such factors is increasingly underscored in more recent publications.[107-108]

In developing countries, for example, the use of kerosene, gas, coal, liquefied petroleum gas, straw or wood for heating and cooking reportedly contributes to elevated levels of certain indoor air constituents, and may lead to increases in disease incidence.[109-110] Early, limited reports first suggested that exposures to indoor air constituents may be responsible for the increased risk of lung cancer among Oriental women.[111-112] Since then, a number of studies have reported elevated risks for lung cancer associated with the use of coal in stoves and other indigenous heating devices in China.[47,63-65,113-118] At least two papers have reported that certain cooking techniques which produce oil vapors, e.g., stir-frying and deep frying, were associated

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with increased lung cancer risks among Chinese women.[63,119] Another paper reported that national air quality standards were consistently exceeded in the Chinese kitchens studied.[120] Similarly, the use of kerosene, coal, wood or straw has been associated with an increased risk of lung cancer in Japanese women.[46,61] It appears that poor indoor air quality may not be restricted to Asian countries; similar observations regarding workplaces and offices have been made in Greece as well.[121]

In addition to poor indoor air quality from heating and cooking fuels, at least one epidemiologic study from northeast China reported statistically significant associations between outdoor air pollution and lung cancer incidence.[118]

Several aspects of diet have been suggested to be possible confounding factors. For instance, Dr. Linda Koo, in several papers published between 1988 and 1990, re-analyzed her data on nonsmoking wives of smokers.[122-125] Her results indicated that wives of nonsmoking husbands had "healthier" lifestyles than wives of smoking husbands; they exhibited higher socio-economic status and had better indices of family cohesiveness and lower frequencies of selected health problems and complaints. An important and statistically significant difference was reportedly found in the diets of the two groups. Wives of smoking husbands consumed more processed and spicy foods and ate fewer fresh fruits and vegetables than

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wives of nonsmoking men. Koo concluded that such correlates of smoking status "act as important confounders when evaluating health risks among families with smoking husbands." She also wrote:

> [C]aution should be exercised when interpreting data on ETS. It may not be the hazards of tobacco smoke that are being evaluated, but a whole range of behaviors that result from having a smoking husband, which may in turn increase the risk for certain diseases among their wives and children.[122]

Moreover, in 1989, Sidney and colleagues reported that nonsmokers living with smokers consumed less carotene (Vitamin A) than nonsmokers who lived with other nonsmokers. They concluded that "dietary beta-carotene intake is a potential confounder and should be measured whenever possible in studies of the relation between passive smoking and lung cancer."[126] In 1991, additional supportive data were provided by Le Marchand and colleagues' study of women in Hawaii, which found that beta-carotene intake was inversely associated with ETS exposure.[127] The authors noted: "Decreased beta-carotene consumption could explain, in part or in total, the moderately elevated lung-cancer risk observed for passive smokers in past studies." In addition, in a 1990 abstract, Waller and Smith reported a correlation between serum beta-carotene level and socioeconomic status, suggesting that the relationship may be even more complex.[128] Another aspect of diet, namely, consumption of green tea, was associated with a statistically significant

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increased risk of lung cancer in women in Hong Kong.[129] Similar results were first reported at a 1987 conference in Tokyo, in a paper calling for increased attention to possible relationships between substances taken in orally and lung cancer.[130]

Other potential confounders in studies of ETS and lung cancer include occupational exposures;[46-47,63,118,131] personal health factors (e.g., menstrual cycle length, history of respiratory disease);[47,63,65,121] family history of lung cancer;[63,65] and even keeping pet birds.[132]

Swedish scientist Ragnar Rylander summed up the importance of confounders in a 1990 article.[108] He cautioned that studies evaluating the relationship between exposure to ETS and lung cancer "must take into account other environmental risk or protection factors and the possibility that exposure to environmental tobacco smoke may be confounded," which he noted "has not been considered in the majority of such studies." He concluded: "Until this has been done, the claim of causality between environmental tobacco smoke and lung cancer remains uncertain."

Mathematical Models

In 1985, Repace and Lowrey, two antismoking advocates, published an article in which they estimated that exposure to ETS

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is responsible for 500 to 5,000 lung cancer deaths a year among nonsmokers in the United States.[133] The risk estimates were cited widely in the press prior to the article's publication, perhaps because it was incorrectly identified as a study by the EPA. In reality, the article was neither approved nor endorsed by that agency.

The article itself presents two highly theoretical models for estimating risks of lung cancer from ETS exposures. The first model relies upon a "reinterpretation" of the epidemiological studies of lung cancer in nonsmokers, and the second provides an estimate for lung cancer mortality derived from data in a study of Seventh Day Adventists, a religious group known for its vigorous opposition to smoking.

Critics of the latter approach have pointed out that the estimates are based on errors and "unrealistic assumptions" which result in overestimations of exposure.[134-135] For example, one recent analysis of the Seventh Day Adventist model showed that, depending upon the assumptions and data used, such model estimates are inherently inaccurate and may vary by as much as 300-fold.[136] Another scientist noted that the exposure and dose levels they used are not based on actual measurements, and that actual measurements reported by other researchers range from "ten-to-one-hundred-fold less than that in the Repace and Lowrey

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model."[137] Still other scientists have questioned both methods of analysis used in their article.[138-140] They suggest that Repace and Lowrey failed to control for other confounding factors, and that their model did not provide "the very statistical bases of estimation procedures."[137]

Repace and Lowrey's estimate of nonsmoker lung cancer risks in the workplace has been criticized by another group of scientists who pointed out that none of the epidemiological studies which examined the relationship between ETS exposure in the workplace and disease in nonsmoking women report a statistically significant increase in risk.[134] Repace and Lowrey have failed to address these criticisms, and, in fact, published a similar article in 1990, in which they conclude that 5,000 (plus or minus 2,400) lung cancer deaths are due to ETS each year.[141]

In 1988, British researchers Darby and Pike published a paper describing another type of mathematical model which purportedly can be used to predict potential effects from ETS exposure based on data from a study of active smoking.[142] However, even after adjusting for childhood exposure to ETS, the authors noted that the predicted risk for nonsmokers was smaller than "the underlying background risk of lung cancer," and that their model could not explain the difference between risks reported for nonsmokers in epidemiological studies and the low levels of ETS

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exposure reported in other types of studies. When the validity of their model was challenged,[143] they replied that the existing data for the indicator of ETS exposure they utilized were insufficient and inappropriate for making comparisons between the epidemiologic and extrapolation approaches to risk assessment.[144]

Summary

Attempts to generate statistically significant results from epidemiological studies on ETS and nonsmoker lung cancer are unconvincing, due to scientific deficiencies in each of the studies.[145] A German specialist in biometrics and epidemiology, in his assessment of these studies, has explained why the data fail to meet the criteria which some regard as necessary to establish a causal relationship:

> The majority of criteria for a causal connection are not fulfilled. There is no consistency, there is a weak association, there is no specificity, the dose-effect relation can be viewed controversially, bias and confounding are not adequately excluded, there is no intervention study, significance is only present under special conditions and the biologic plausibility can be judged controversially.[145]

Given these difficulties in interpretation, it is therefore not surprising that an eminent statistician has observed:

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> [I]t is unlikely that any epidemiological study has been, or can be, conducted which could permit establishing that the risk of lung cancer has been raised by passive smoking. Whether or not the risk is raised remains to be taken as a matter of faith according to one's choice.[102]

This same statistician concluded: "What with subtle biases, not so subtle biases, and even extravagant errors, one should not accept too readily claimed demonstrations of ill effects of passive smoking."[72]

Overall Mortality and Cancer Other Than the Lung

A few articles have claimed that the risk for all cancers is higher among nonsmokers exposed to ETS. One such study by Miller examined a population which was selected from death notices in a northeastern U.S. newspaper.[146-147] However, analysis of the data reveals that the reported risk disappears when age comparisons of cases and controls are considered.[148]

In a series of papers based on data of cancer cases selected from a North Carolina hospital tumor registry, Sandler, et al., reported increased risks for lung cancer and cancer of other sites in nonsmokers exposed to ETS.[149-151] Yet, published criticisms cast serious doubt on these conclusions. One commentator challenged the authors' selection of controls and their use of

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questionnaire information.[152] Another scientist observed that under one interpretation, the data suggest that exposure to ETS had no effect on overall cancer incidence.[153-154] A third critic called the studies "seriously flawed" and questioned the authors' methods of data collection.[155] Still others challenged their conclusions because the reports had not considered potential confounding variables such as diet, hygiene and lifestyle.[156-157]

In 1989, Sandler, et al., evaluated mortality from all causes for nonsmokers in a Maryland county. Although the authors reported slightly higher risks for nonsmokers who lived with smokers than for those who lived with nonsmokers, they acknowledged that "these small nonspecific increases in death rates may reflect other characteristics of passive smokers that increase mortality."[158] This paper was also strongly criticized by a reviewer who wrote that it should have been rejected for publication "on the basis that it is poor science and inadequately designed and undertaken to address the issue presented in the title."[159]

A. Judson Wells, in a 1988 publication, estimated that in the United States thousands of nonsmoker deaths from all causes could be attributed to exposure to ETS.[160] Yet even the editor of the journal which published his paper reported that one pre-publication reviewer had recommended that the paper not be published because it was "too speculative."[161] Three critical responses were

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published in 1990,[107,162-163] one of which called Wells' estimates "scientifically unjustified."[107] However, Repace and Lowrey praised Wells' work, and Wells himself attacked the three critical commenters, although he failed to address the issues they raised.[164-165]

With regard to the issue of ETS and overall mortality, Vandenbroucke and colleagues' 25-year follow-up study of over 1,000 married couples in Holland reported that "passive smoking was not associated with an increase in total mortality."[166] Although the authors cautioned that the results do not necessarily contradict those which suggest such an association, they added that their findings "were reassuring to the extent that the possible absolute risk carried by passive smoking is probably small."

Only a few studies have considered ETS exposure and cancer of individual, non-pulmonary, sites. In 1989, Slattery and colleagues reported an increased risk of cervical cancer in nonsmoking women who reported three or more hours of ETS exposure per day.[167-168] However, the study was strongly criticized for its failure to account for all pertinent confounding variables.[169] Kabat, et al., and Burch, et al., have independently investigated bladder cancer incidence and ETS exposure;[170-171] neither study reported an association between bladder cancer and exposure to ETS at home or at work.

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Two studies, by John, et al., and Grufferman, et al., have suggested that there may be an association between childhood ETS exposure (particularly paternal smoking) and the incidence of certain childhood cancers; however, both studies involved very small numbers of cases.[172-173] Finally, isolated reports have claimed that their data support an association between ETS exposure and brain and nasal sinus cancers,[1] or breast cancer.[1,149] Without adequate replication of these results, it is not possible to reach any definitive conclusion about ETS exposure and non-pulmonary cancer incidence.

A presenter at a 1989 scientific symposium in Montreal, Canada, who had reviewed many of these studies, contended that the studies available at that time provided "insufficient data to evaluate the effect of ETS on cancer other than of the lung."[174] He also asserted that the studies had numerous shortcomings: "The reported associations are weak and inconsistent, and are subject to the potential effects of biases and uncontrolled confounding factors . . . ."

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Lung Function/Disease: Adults

The issue of a possible relationship between exposure to ETS and respiratory disease in nonsmokers was first raised in a 1980 study by two California researchers, White and Froeb.[1] On the basis of their measurements of the small airways function of smokers and nonsmokers in the workplace, they asserted that nonsmokers exposed to tobacco smoke at work for 20 or more years had reduced function of the small airways compared to nonsmokers who did not have such exposures. Other studies with similar findings have been reported since that time. In 1983, French researchers Kauffmann, et al., reported that nonsmoking spouses over 40 years of age who were married to smokers exhibited decreases in pulmonary function compared to nonsmoking spouses of nonsmokers.[2] A 1989 study by Hole, et al., of a Scottish population reported significantly lower pulmonary function among nonsmokers who lived with smokers compared to nonsmokers who were not exposed to ETS.[3]

However, each of these studies, particularly the White/Froeb study,[4-7] has been criticized for numerous reasons. In regard to the White/Froeb study, for example, a physician at a U.S. medical school questioned their use of carbon monoxide as an index of smoke exposure, contending that they "do not have reliable estimates of the smoke exposure in the environment of their nonsmokers" because it is not unique to tobacco smoke.[7] A British

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reviewer shared the physician's view that their findings "relate to an index which is contentious and certainly not an accepted reliable indicator of an increased health risk."[8] White and Froeb themselves noted that the average values of the pulmonary tests of nonsmokers exposed to tobacco smoke "were not notably different" from the values suggested as normal by a specialist in this area.[9]

Perhaps the most telling criticisms of the study were voiced by Dr. Michael Lebowitz of the University of Arizona at an annual joint meeting of the American Lung Association/American Thoracic Society and in a subsequent letter published in the U.S. Congressional Record.[10-11] During a forum at the ALA meeting, Dr. Lebowitz stated that he had concluded, from his own extensive review of the study and from an interview with White, that the study was "improperly designed" from an epidemiological point of view. He noted that there were problems "inherent" in the study, including the selection of the study group and the measurement of smoke in the workplace. Dr. Lebowitz also expressed concern that the statistical significance of the data appeared to depend on the unexplained omission of data for 3,000 people who were originally included in the study. Based upon these considerations, Dr. Lebowitz urged that the study not be used to support the claim that ETS affects the lung function of adults in the workplace.

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Dr. Lebowitz again took issue with the White/Froeb study in 1984 in a paper he presented at the Vienna Symposium on Passive Smoking. He contended:

> 'Even with a biased population, poor study design, and incorrect statistical evaluation, there were no clear-cut, consistent, medically meaningful differences between passive smokers and groups of nonsmokers; a corrected statistical analysis strengthened this conclusion.[12]

In the same paper, Dr. Lebowitz also questioned the findings of the 1983 French study which reportedly found significant differences in lung function between exposed and nonexposed nonsmokers in one part of the study population but no significant differences in the population as a whole. Dr. Lebowitz noted that since the "healthiest" part of the study population lived in the most polluted areas, the study may have been flawed due to biased population selection and testing or other confounding factors.[12]

In addition to these concerns, the results of the White/Froeb, the French and the Scottish studies appear to be inconsistent with other research on lung disease and lung function in nonsmokers. For example, a 1984 study of 1,351 German office workers by Kentner, et al., reportedly found no effect of ETS on pulmonary function among exposed nonsmokers.[13] In a 1988 update

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of the study, the investigators noted that "there is no evidence that average everyday passive smoke exposure in the office or at home leads to an essential reduction of lung function in healthy adults."[14] The key investigator for the study reported these same findings in 1989 and 1990 publications.[15,16]

Similarly, Canadian researchers Shephard, et al., reported that their analysis of a group of healthy young adult nonsmokers showed "no consistent reaction" of static lung volumes and insignificant changes of other lung function measurements when they were exposed to tobacco smoke.[17]

Numerous studies of individuals exposed to ETS in the home also present conclusions which appear to be contrary to these three studies. In 1981, Comstock, et al., epidemiologists at the Johns Hopkins University School of Medicine in New York, reported that in a group of 1,724 adults, the frequency of respiratory symptoms in nonsmokers was <u>not</u> significantly associated with the number of smokers in the household.[18] Nor was the frequency of impaired ventilatory function significantly higher if there were smokers in the home. However, their analysis did show that "among men who never smoked cigarettes, gas cooking was definitely associated with impaired ventilatory function, even when corrected for multiple comparisons."

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Other studies on pulmonary function, respiratory disease and environmental tobacco smoke deserve special mention. In 1983, for example, Jones, et al., reported that in a case-control study of several hundred nonsmoking women from a U.S. study population, there was no significant association between decreases in lung function and exposure to ETS in the home.[19] Similarly, Lebowitz, et al., coordinators of an epidemiologic study of obstructive lung disease in Arizona, reported finding no effects from ETS exposures in the home in their adult study population.[20] In a study of 376 families, Yale University scientists Schilling, et al., also reportedly found no evidence that environmental tobacco smoke affects either lung function or respiratory symptoms in adults.[21]

More recently, in a Canadian study on the effect of indoor and outdoor pollutants on the lung function of housewives, Hosein and Corey concluded that ETS exposure did not significantly influence lung function, but that gas stove usage played a role in average lung function decline.[22] These findings have been reported by others. For example, a Japanese research group, Nitta, et al., has also reported that gas stove usage affects the respiratory health of adults.[23] Koo and Ho recently studied the effects of indoor nitrogen dioxide (NO_2) levels on respiratory illness rates in Hong Kong.[24] They reported that indoor NO_2 levels were associated with allergic rhinitis and chronic bronchitis in non-

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smoking mothers, but that there was no relationship between environmental tobacco smoke and NO_2 levels in the home environment.

In 1990, a Greek research group, Kalandidi, et al., investigated the possible relationship between reported tobacco smoke exposure and chronic obstructive pulmonary disease (COPD) in adult nonsmokers.[25] Although they reported finding increased risks of COPD among nonsmokers reportedly exposed to ETS, it is not clear whether they adequately considered other potential exposures which have been associated with the development of COPD such as occupation and outdoor air pollution. Another study published in 1990 by Lebowitz and Quackenboss reported that while they observed an increase in several acute respiratory symptoms associated with exposure to ETS, they noted "no relationship between the prevalence rates for chronic symptoms and diseases and the presence or amount of ETS in the home."[26]

In 1990, Jedrychowski, et al., reported an increased occurrence of respiratory illness among a group of elderly women exposed to ETS, but conceded that the reported effects were "greatly confounded by other indoor pollutants, especially those associated with cooking the meals on gas ovens." They also conceded that they considered only "the current passive smoking experience at home and took no account of the history of passive smoking that

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might have been present in the work place," which could "evidently bias the results of the study with respect to passive smoking."[27]

Researchers who have analyzed these reported findings contend that the results are mixed and inconclusive. For example, researchers in France, Laurent, et al., have commented that purported long-term health effects from exposure to ETS are difficult to demonstrate in healthy adults and that the results of the epidemiological studies are "sometimes conflicting and often open to question."[28] One investigator who is critical of ETS conceded that "the effect of passive smoking on respiratory infections in adults has not been well characterized and reports of its effects on chronic respiratory disease in adults have been inconsistent."[29]

In his summary of the studies of respiratory symptoms and disease in adults published up to early 1990, American investigator Dr. Philip Witorsch noted that "4 of 8 [reported] an increased frequency, 4 of 8 no increased frequency."[30] He stated that, in addition to "all of the problems" with the studies, "these results are too variable to permit any conclusion of association." As his summary demonstrates, the conclusion of the participants at the 1983 U.S. National Institutes of Health workshop on ETS exposure, that the possible effect from ETS "varies from negligible to quite small,"[31] is still pertinent.

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"Compromised Individuals": Adults

Asthmatics are believed to be particularly vulnerable to various environmental influences, including ETS, but the scientific data in this area are contradictory and inconclusive. A 1981 study by Dahms, et al., for example, reported decreases in the pulmonary function of several asthmatics exposed to environmental tobacco smoke.[1] However, the study suffers from several obvious limitations, including the unrealistic conditions under which the subjects were exposed to ETS; they were challenged with high levels of smoke in an enclosed smog chamber. In addition, as the authors themselves noted, their experiment lacked proper controls and the effects observed may have been due to psychological, not physical, factors.

Contrary to Dahms, et al., a Canadian group, Pimm, et al., observed no systematic lung changes among asthmatics exposed to levels of tobacco smoke typically found in public places.[2] Later research by the co-authors of this study supports this conclusion. They reported that respiratory data collected from a group of asthmatic volunteers exposed to tobacco smoke "do not suggest that asthmatic subjects have an unusual sensitivity" to such exposure.[3] Although several volunteers claimed to have experienced wheezing and tightness in the chest due to the exposure, the researchers determined that the "physiological data give little

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support to the concept of a subgroup with particular sensitivity." They noted that these reactions probably were due to the "suggestibility" of the subjects.

The difficulty in determining the impact of psychological responses in such studies is clearly demonstrated by the results of two reports from Australia. Although a 1985 report by Knight and Breslin suggests that short-term ETS exposures are capable of inducing reactions in asthmatics,[4] an earlier study co-authored by Breslin tends to support the theory that psychological reactions may partially explain symptoms observed during such exposures. In that study, Breslin and Ing reported that although asthmatics exposed to tobacco smoke complained of subjective symptoms, no significant objective evidence of airways obstruction was observed.[5]

Other studies also fail to provide support for claims regarding a relationship between ETS exposure and reactions in asthmatics. For example, Wiedemann, et al., at Yale University, examined the short-term effects of ETS exposure on a group of young asthmatic patients and observed no changes in lung flow rates.[6] They concluded that such exposures present "no acute respiratory risk" to asthmatics.

Tulane University scientists Stankus, et al., recently assessed the effects of heavy exposure to ETS in a group of self-

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reported "tobacco smoke-sensitive" asthmatics. They reported that two-thirds of the subjects did not experience significant changes in pulmonary function even after heavy, prolonged exposure to ETS, and that there was no association between reactions to ETS and hypersensitivity to tobacco leaf extract, which is commonly used in allergy testing.[7]

A report by Lebowitz on data from a large-scale epidemiological study in the U.S. suggests that ETS in the home does not affect symptoms or pulmonary function in either children or adult asthmatics, but that dust and pollen in the home apparently can provoke such effects.[8] Another group of U.S. researchers, Bailey, et al., also recently reported that exposure to ETS did not impair lung function in 263 asthmatic adult subjects.[9]

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Lung Function/Disease: Children

Perhaps no claim regarding ETS is as capable of provoking strong feelings as the charge that parents who smoke may harm the health of their children. While the issue of parental smoking is laden with emotion, the scientific basis for the claim is difficult to interpret. For example, while one study examines respiratory symptoms or illness such as coughs and colds by questionnaire responses from parents,[1] another measures lung function with special equipment at a school or health facility.[2] In the U.S. alone, according to one report, this has led to a situation in which studies of ETS and the respiratory system are "being carried out by at least three different groups, are employing different populations and methodologies and have led to varying conclusions."[3]

Perhaps not surprisingly, such studies, each with a different sample size, data collection method, and analysis, tend to yield factually incompatible and contrary conclusions. For instance, although certain studies have reported adverse findings between parental smoking and respiratory illness in children,[4-35] others have observed no significant relationship.[1,36-51] After a five-year study of over 400 children, for example, a Dutch research group concluded there was "no evidence" that parental smoking had an appreciable effect on respiratory symptoms in school children.[43]

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A similar conclusion was reached by a group of U.S. researchers, including a critic of smoking, who found "no significant relation" between parental smoking and respiratory symptoms in a study of nearly 400 families with 816 children in three cities.[36]

The contradictory nature of findings on the issue of parental smoking is also apparent in the growing number of studies examining the relationship between parental smoking and children's respiratory or lung function. Although several reports have claimed that parental smoking results in decreased pulmonary function in children,[8,13-14,32,52-60] others have not,[61-64] including those of a U.S. research group who have published a series of studies on this subject.[2,65-67] In 1982, for example, the U.S. group showed that a comparison of body size with lung function eliminated any reported correlation between parental smoking and children's lung function.[2] Two years later, a reanalysis of data from families in their study population again showed that "parental smoking did not have a significant effect on children's pulmonary function; smoking habits of others in the household (predominantly siblings) did not have any effect either."[66]

Moreover, the authors of studies reporting adverse effects from ETS exposures among children concede that their conclusions must be viewed with caution because of numerous confounding factors. The potential impact of such factors was given special consideration

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in the report from a workshop on ETS sponsored by the U.S. National Institutes of Health.[3] After listing numerous such factors, including types of heating used, socio-economic status and demographic and medical characteristics of the study population, the report cautioned "that any study which ignores them will be seriously flawed."

The importance of such factors in evaluating the outcome of research on parental smoking is supported by a number of reports which have shown that the use of gas stoves in the home may be independently associated with respiratory disease[40,65,68-72] and impaired pulmonary performance[73-74] in children. One group of British researchers acknowledged the possible influence of factors such as cross-infection in the home and genetic susceptibility to childhood respiratory illness and symptoms.[11-12] More recently, researchers in Hong Kong reported "a highly significant correlation" between the frequency of respiratory illnesses of mothers and their children.[75]

Other confounding factors independent of parental smoking have been reported recently in the literature. For example, studies in the United Kingdom have identified damp housing[46, 76-79] and paternal occupation[80] as potential explanatory factors for the occurrence of respiratory illness in children. Other recent studies have identified outdoor air pollution,[82-85] infections

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transmitted during day-care attendance[49,86-87], decreased breast-feeding[88] and the use of kerosene heaters and woodburning stoves in the home[89-91] as factors related to childhood respiratory disease.

The relevance of dampness in the etiology of respiratory symptoms in children is supported by current research which links dampness with the presence of molds, dust mites, fungi and other allergenic microbes. The growth of fungi and molds in the home is directly related to respiratory symptoms and sensitization reactions in some individuals.[78,92-94] Recent investigations report, moreover, that exposure to ETS does not increase sensitization to common allergens in children.[95-96]

Others have conceded that the reliance of such studies on questionnaires for information about respiratory symptoms casts doubt on their reported findings. In one study that reported a significant association between parental smoking and respiratory symptoms, for example, it was noted that even "slight changes" in the way the questions were phrased could result "in substantial differences in the type of responses one obtains."[14] Similarly, another study observed that there was a significant difference in the respiratory symptoms reported depending on which parent completed the questionnaire.[24] Authors of another study that reported adverse effects of parental smoking on the respiratory

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health of children conceded that "since the exposure variables used in these analyses were subject to substantial measurement error, a more refined measurement of personal exposure is required."[29] One researcher who is critical of parental smoking has stated that "quantitative assessment of involuntary exposure of infants and children to ETS has been very imprecise and probably inaccurate."[97]

Studies utilizing seemingly more objective standards such as actual measurements of lung function are also open to criticism. Even reviewers of the literature who are critical of parental smoking concede that the tests used in these studies are "influenced by a large number of variables."[98] They list age, height, and gender of the test subject as well as his or her motivation, cooperation, and effort put forth during the test, the skill and experience of the operator, and the type of instrumentation used as variables that can affect the results of pulmonary function measurements. The reviewers explain that these problems are especially important in pulmonary function measures taken in children. In 1989, two American co-researchers, Witorsch and Witorsch, reported that "it has been shown that mean pulmonary performance within a single group of children can vary significantly from one spirometry test to the next without any apparent cause" and that it "is noteworthy that such statistically significant

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differences are similar in magnitude to most of the small decrements in pulmonary function reported in children of smoking parents."[99]

The shortcomings of studies analyzing the relationship between ETS exposure and childhood health were highlighted in a 1988 report by two U.S. investigators who re-examined 30 such studies and evaluated them for their scientific validity.[100] They noted that while several studies of adequate scientific design had reported a statistically significant relationship between ETS exposures and childhood health, "most studies had significant design problems that prevent reliance on their conclusions." The authors concluded that "many questions remain, and future studies should consider important methodological standards to determine more accurately the effect of passive smoking on child health."

Thus, claims that parental smoking plays a causal role in the development of respiratory symptoms and reduced lung function in children are not scientifically justified. Such claims are typically based upon a single study of a selected symptom (such as cough or wheeze). These kinds of studies invariably fail to consider nutrition, health habits of the family, and other lifestyle variables. Similarly, studies that report reduced lung function in children of smoking parents fail to address the issue of socio-economic status or the potential role of genetic and family traits in pulmonary function capabilities.[64] Moreover, the reductions

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reported in the literature are small and of uncertain clinical or biological significance, and are contradicted by a number of studies that reportedly have observed no effect of parental smoking on children's lung function.

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"Compromised Individuals": Children

As with studies of adults who have chronic lung problems, the data on parental smoking and its possible effects on children with lung conditions are inconclusive.[1-9] In 1990, German researchers Oldigs, et al., reported that exposing children with bronchial asthma to cigarette smoke sufficient to reach a level of 20 parts per million (ppm) carbon monoxide for one hour did not affect their lung function or bronchial responsiveness.[8] Similarly, Charles Sherman and his co-investigators reported that neither paternal nor maternal smoking "bore an apparent relation to the development of asthma" in a sample of children, aged 5 to 9, enrolled in public and parochial schools in East Boston, Massachusetts in 1974.[9]

A British researcher, Strachan, has implicated damp housing as a potential factor in the development of childhood asthma.[10] After controlling for the possible influence of housing tenure, number of people per room, number of smokers in the household, and gas cooking, he reported that the relationship between damp housing and childhood asthma in his study population remained "highly significant."

Other investigators, Rubin and Gilljam, et al., have suggested that parental smoking might worsen the condition of

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children with cystic fibrosis, a congenital disease usually developing during childhood and causing pulmonary disorders.[11-12] However, these studies are subject to the same uncertainties exemplified by other studies on parental smoking. For example, although Rubin reported an association between parental smoking and decreased nutritional status and growth of children with cystic fibrosis, he conceded that "the possibility cannot be ruled out that social, economic, or other factors determined both the smoking status of the household and the nutritional status of the child."[11]

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Heart Disease

Assertions regarding a relationship between heart disease and ETS exposure are primarily based on a small number of epidemiological studies which suffer from a variety of serious methodological weaknesses. In total, 11 epidemiological studies have reported data on a possible statistical association between ETS and heart disease incidence or mortality.[1-11] However, nearly two-thirds of these studies -- seven of the 11 -- failed to report a statistically significant association. Of the four studies that claimed to find a statistically significant relationship,[3-5,9] three are very small-scale,[3-4,9] which means that their conclusions are based on only a few cases and, therefore, may not be very reliable. All of these studies suffer from numerous methodological weaknesses, including small sample sizes, potential misclassification of the smoking status of study participants, inadequate assessment of ETS exposure, failure to control adequately for biases stemming from potential confounding variables and failure to confirm causes of death via autopsy or other histological methods.

Despite these scientific weaknesses, several recent literature reviews have concluded that ETS is associated with an increased risk of heart disease and that, in fact, such exposure causes a large number of deaths among ETS exposed nonsmokers each

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year.[12-16] Four of these reviews attempted to make a specific estimate of overall risk based on the combined data from the epidemiologic studies.[12-15] The authors of these reviews generally agreed that ETS exposure is associated with an approximately 30 percent increase in heart disease risk (risk ratio of 1.3). The most recent, and certainly the most widely publicized, of these reviews was written by Stanton Glantz and William Parmley of the University of California, San Francisco.[15] They argued that their estimate of a 30 percent increase in heart disease risk in ETS-exposed nonsmokers translates into 37,000 excess heart disease deaths in the U.S. each year.

The usual basis for overall risk estimates, including the one by Glantz and Parmley, is meta-analysis. This technique is appropriately used only when the underlying studies are very similar and of high quality.[17] In the case of ETS and heart disease, however, the risk estimates are based on the meta-analysis of a small number of epidemiological studies that are highly dissimilar in terms of the methodology used, the study populations and the disease endpoints. Furthermore, the individual studies often suffer from serious methodological and other scientific weaknesses. For these reasons, the validity of any meta-analysis based on these data is highly questionable.

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Claims of an association between ETS and heart disease often receive a great deal of publicity. However, there is a serious scientific debate about the basis for such claims. This is reflected in the wide range of reviews by a variety of individuals from several countries and perspectives who have argued that the available data do not allow any conclusions concerning the question of ETS and heart disease.

The earliest reviews dealing with this question appeared even before much of the epidemiological data became available in the early 1980s.[18-20] While they emphasized the near absence of epidemiological studies on ETS and heart disease, they did discuss data available concerning carbon monoxide (CO) and nicotine, which are often claimed to be the primary substances in ETS that lead to cardiovascular damage. The most extensive of these discussions was a 1984 review by two German researchers, who concluded that, at the levels reported for ETS-exposed nonsmokers, "neither CO nor nicotine is likely to play a role in the development and progression of coronary heart disease."[20]

The first major reviews of the epidemiological data on ETS and heart disease appeared in 1986. In that year, the report from the Office of the U.S. Surgeon General examined the available data and judged that "no firm conclusion" could be made regarding a possible relationship between ETS and heart disease.[21] Also in

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1986, the NAS Report stated that any potential heart disease risk related to ETS would be "difficult to detect or estimate reliably" from epidemiological studies, and would be "the same order of magnitude as what might arise from expected residual confounding due to unmeasured covariates."[22]

As additional epidemiological data on ETS and heart disease have been published, subsequent evaluations have often reinforced the positions of these two reports. There are many examples. In 1988, a review from two scientists at the University of California - Los Angeles, which generally argued that ETS is associated with a disease risk in nonsmokers, nevertheless commented that for heart disease, "no firm conclusion that a causal relation exists is yet warranted."[23] A similar view was expressed in a 1988 review from a Harvard physician, who said that there were "no clear data" that ETS increases heart disease risk.[24] Both of these reviews called for more research.

Following a detailed discussion of the recent epidemiological studies on ETS and heart disease at the ETS conference held at McGill University, a New York Medical College faculty member concluded that "none" of the studies "provides any basis for altering the Surgeon General's and NAS's conclusions concerning ETS and cardiovascular disease."[25]

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Several additional evaluations of the literature on ETS and heart disease appeared in 1990. Two of these were reported at international conferences in Lisbon, Portugal[26] and in Budapest, Hungary.[27] Both argued strongly that the data on ETS and heart disease were methodologically weak and insufficient to draw conclusions. A third, by a physician from a hospital and medical school in Dundee, Scotland, was in the context of a broad review of many factors potentially involved in heart disease.[28] Although he argued that smoking cigarettes is a risk factor and cause of death among smokers, he nevertheless suggested that attempts to attribute heart disease deaths to ETS exposure "may be pushing epidemiologic evidence to the limits of what it can show." In another major review, this one from the United States, two physicians concluded that "the data that are available are so sparse that any attempt to reach a definitive assessment would be fraught with uncertainty."[29]

Perhaps the most recent review of the literature on ETS and heart disease was published in 1991 by the former director of toxicology of a major European research laboratory. He described the scientific data relating to a possible heart disease risk in nonsmokers exposed to ETS as "not very convincing."[30]

Several evaluations of the literature on the ETS and heart disease have been prepared under the auspices of a number of

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national governments. As with the 1986 U.S. Surgeon General's Report,[21] they often voice strong opinions that smoking and ETS exposure are associated with serious health risks. Nevertheless, they argue that the data on ETS as a possible cause of heart disease are inconclusive. For example, in 1987, reports dealing with ETS were issued by Canada's Minister of National Health and Welfare[31] and by Australia's National Health and Medical Research Council.[32] Both reports described the data as "limited" and inadequate to permit a conclusion to be drawn.

European government reports have also expressed similar concessionary opinions concerning ETS and heart disease. In 1990, a Committee of the Health Council of the Netherlands prepared a report at the request of the Dutch government. The Committee judged that the "currently available data preclude a firm conclusion as to whether exposure to tobacco smoke is a contributory factor in the onset of and mortality from cardiovascular disease in nonsmokers."[33] In Norway, a Report from a Working Group appointed by the Norwegian Directorate of Health also examined this issue. Although the report acknowledged there are data suggesting a statistical association between ETS and heart disease, it judged that "no definite causal connection can be ascertained at the present."[34]

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Heart Disease: Experimental/Biochemical Studies

In addition to the epidemiological studies, there are several experimental and biochemical studies that have been cited as supporting an increase in heart disease risk stemming from ETS exposure. A few of these reports claim that ETS exposure adversely affects exercise capacity[35-37] and that, in the case of heart disease patients, this can lead to attacks of angina (heart pain).[36-37] Other reports have attempted to demonstrate that exposure to ETS adversely affects some aspect of cardiovascular function, particularly blood clotting[38-41] and cholesterol levels.[42-43]

In the area of exercise performance, three studies are generally cited. In one of these, a 1985 report by McMurray, et al., ETS exposure was claimed to adversely affect the exercise performance of healthy subjects.[35] In the two other studies, one by Aronow[36] and the other by Khalfen and Klochkov,[37] heart disease patients exposed to ETS reportedly were unable to exercise as long before experiencing angina. The credibility of the Aronow report, as well as other research by this investigator, has been widely challenged.[44-47] The Khalfen and Klochkov report is a Russian language article about which relatively little is known. Regarding any of these studies, whether conducted with healthy subjects or heart disease patients, the general criticism can be made that it

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is almost impossible to "blind" either the experimenter or the subjects to ETS exposure. Thus, there is always the possibility that some subjective psychological factor may have influenced the results.

There are very limited data attempting to demonstrate that ETS adversely affects some process that might be involved in blood clotting or atherosclerosis. The hypothesis of the studies from which these data are derived has been that ETS may increase the tendency of certain blood components, known as platelets, to stick together. This claim has been made based mainly on data in four published reports,[38-41] three of which are from the same Austrian research group.[38-40] The fourth report, from a group of researchers in Kansas City, Missouri,[41] suffers from serious methodological weaknesses, particularly its failure to establish a proper control for comparison purposes.

Finally, there are two reports which assessed cholesterol and other blood components among children in relation to parental smoking status.[42-43] Both reports claim that parental smoking is associated with decreases in HDL (high density lipoprotein) cholesterol, which some have argued may be associated with heart disease risk. These studies measured components of blood as the endpoint, but are essentially epidemiological studies in that they, at best, may suggest statistical associations. As such, they suffer

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from weaknesses characteristic of other epidemiological studies of ETS exposure, especially difficulties in controlling for potential confounding variables and inadequate assessment of ETS exposure. Furthermore, the potential significance of blood values in relation to later heart disease risk in a group of children is highly speculative.

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Other Children's Diseases and Conditions/Pregnancy

There are studies which claim to have found an association between exposure to ETS and the occurrence of a relatively common childhood ear condition called otitis media with effusion (OME) and the incidence of low birth weight infants.[1-3] However, as with other claims regarding ETS, the reported data are inconsistent, even contradictory, in nature.

For example, in regard to OME, an inflammation of the inner ear that can lead to the effusion or accumulation of fluid in the inner ear canal, a group of Dutch researchers has asserted that "there is little evidence that parental smoking has an effect on the risk for OME," although they noted that "the literature is not consistent."[4] Their own study indicated that while the occurrence of OME was not related to exposure to ETS in the home, variables relating to age, season, family size, sibling's history of OM, frequent swimming, and public day care attendance had a "significant effect." A Scottish study which did report an association between parental smoking and OME noted that the prevalence of parental smoking was higher in rented or crowded homes, and in homes affected by dampness or mould growth.[2] Clearly, these reports suggest there is a need to evaluate additional factors in any study of the relationship between OME and parental smoking.

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Other researchers recently acknowledged that questionnaire reports of acute OME may be an inadequate method of determining the incidence of the condition in epidemiological studies.[5] Therefore, until a more accurate method of determining the incidence of OME is found, isolating parental smoking as a cause is seemingly unjustified.

In regard to infant birthweight, a 1986 study by Rubin, et al., reported that infants with smoking fathers had lower birthweights compared to those with nonsmoking fathers.[3] However, the results were described as "extraordinarily large" by one reviewer, apparently because the authors failed to control for important confounding variables such as social class.[6] Moreover, a study published in 1987 reported results which contradicted those of Rubin, et al. The authors, MacArthur and Knox, observed that the average weight of infants with smoking fathers was greater than that of infants with non-smoking fathers.[7] Still other studies have reported no significant association between paternal smoking and lower birthweight among newborn infants,[8-11] although one study did report an association with the overall condition of the infant at birth.[10] However, this may be due to some unaccounted for factor. The potential importance of such factors is indicated by a 1990 report of one study which noted that, after adjusting for 57 different confounding factors, paternal smoking had no statistically significant effect on infant birthweight.[11]

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Reports that parental smoking causes otitis media in children and reduces infant birthweight are contradicted by studies reporting no association between parental smoking and OME or infant birthweight. In addition, the methods used in these studies to determine exposure to ETS and the incidence of OME are seemingly inaccurate. Thus, the role, if any, of parental smoking has yet to be determined.

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Allergy

Although some individuals are annoyed by the sight and smell of tobacco smoke and a few even report experiencing irritation, the existence of human allergens in tobacco smoke has not been established scientifically. This is an extremely important point in the ETS debate because those seeking to ban smoking in public places have often done so by claiming that nonsmokers are "allergic" to tobacco smoke.

However, a number of research groups have been unable to conclude that humans actually experience a true tobacco smoke allergy.[1-6] In 1980, for example, a group of researchers noted that "direct evidence that tobacco smoke is immunogenic [capable of evoking a specific response] in man is yet to be documented."[5] A more recent report by this same group affirms this conclusion.[6]

Claims about a human tobacco allergy stem primarily from studies in which tobacco leaf extract has been reported to cause allergic skin responses in some people, usually in those who already experience allergic reactions to other substances such as weeds.[7-9] However, as an English immunologist pointed out, there are "great difficulties" in determining whether positive reactions to tobacco leaf extracts are relevant to clinical responses to tobacco smoke.[4] Although he noted that there may be substances in tobacco smoke

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which could "theoretically" act as such agents, he concluded that "there is no proof that the specific sensitization to tobacco smoke exists."

It has also been hypothesized that tobacco smoke is capable of provoking an asthma attack as an allergic reaction.[10-11] However, a Swedish specialist concluded that such results are not proof of a tobacco allergy because the studies, which used tobacco extracts, did not differentiate between non-specific and true allergic reactions in evaluating the results of skin tests and bronchial provocation.[12] Consequently, he stated, "for the present, the question as to whether allergy to cigarette smoke exists or not should be kept open." Other research affirms this point. Scientists reported in 1988 that tobacco leaf sensitivity was not associated with decreased pulmonary function in allergic asthmatics.[13]

Certainly, there appear to be people who may be sensitive to tobacco smoke, but personal annoyance and emotional reactions should not be confused with genuine allergic reactions. In many cases, the individual may be responding to high room temperatures, lack of ventilation, or even to the mere sight of tobacco smoke. Indeed, Dr. John Salvaggio, the director of an allergic disease center in the U.S., has suggested that reported reactions to tobacco smoke may be irritative rather than allergic. After reviewing the

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studies on the allergy question, he concluded that "there is no proof that tobacco smoke is allergenic in man."[14]

Accordingly, it is not surprising that researchers at the Mayo Clinic failed to find any evidence of tobacco smoke allergies in their tests of subjects who considered themselves allergic to tobacco and tobacco smoke.[15] It has been reported in other studies that people who claim to be "smoke sensitive" did not react more frequently to tobacco leaf or smoke extract than those who are "smoke resistant."[13,16]

On the basis of these data, it must be questioned what people really mean when they say they are "allergic" to tobacco smoke. It may be that they simply do not like the sight and smell of tobacco smoke and are interpreting their reaction to mean that they are "allergic" to environmental tobacco smoke. But, as has been pointed out, such personal reactions should <u>not</u> be regarded as true tobacco smoke allergies.

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Sick Building Syndrome

Advocates of smoking restrictions in the workplace argue that ETS exposure gives rise to a number of complaints, including headaches, nausea, coughs, sore eyes and breathing difficulties. However, research indicates that this complex pattern of symptoms, the so-called "sick building syndrome," is commonly reported in modern office buildings whether or not smokers are present.[1-4]

The reported findings of several Canadian investigators demonstrate this point. In 1983, two of these researchers reported on their extensive review of over 150 indoor air quality evaluations of office buildings compiled by U.S. government agencies, universities and others.[5] After examining the data collected during these evaluations, they concluded that smoking did not significantly affect either indoor atmospheres or the frequency of worker complaints and symptoms:

> The review of available studies does not provide any objective evidence that either pollution levels or patterns of health related complaints differ in some remarkable way between locations with or without smoking restrictions.

They did observe that "inadequate" ventilation creates conditions "where discomfort and illness result irrespective of whether or not smoking is permitted." These observations were reaffirmed in

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reports published in 1987 and 1989.[4,6] In their 1989 report, the researchers noted that smoking was related to complaints in only 12 of 408 (less than three percent) of the building investigations included in the database.

Government investigators with Health and Welfare Canada (HWC), in their report on 94 building studies, also noted that only five percent of the complaints were attributable to indoor constituents such as photocopy machine emissions and ETS.[3] In a 1990 update, these investigators reported on data from a total of 1,362 building investigations. Inadequate ventilation was identified as a problem in 52 percent of the buildings.[7]

Similarly, researchers with the U.S. National Institute of Occupational Safety and Health (NIOSH), in a review of 203 air quality investigations of government and business offices, schools and health care facilities, concluded that tobacco smoke played a contributing role in only four (two percent) of the building complaints investigated.[1] A large majority of the complaints were traced to general building contamination and inadequate ventilation. Recently, NIOSH officials reported on an additional 326 building investigations conducted by the agency through 1988.[8] Over one-half of the investigations revealed inadequate ventilation as the source of complaints.

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In 1988, a representative of a U.S. firm specializing in the maintenance of office air conditioning and heating systems reported on 223 individual indoor air quality investigations of publicly and privately owned office buildings.[2] As in the NIOSH investigations, ETS was implicated in only four percent of the buildings investigated. He stated that the majority of indoor air quality problems in modern office buildings may be traced to inadequate fresh air circulation and to poorly maintained ventilation systems which act as breeding grounds for fungi, bacteria and other contaminants. He also suggested that visible tobacco smoke ought to be considered a symptom, rather than a cause, of general indoor air quality problems, in that ETS is often the only visible sign that a ventilation problem exists.

In 1990, investigators from the same firm reported on a survey of 26 commercial office buildings in 20 cities in Switzerland.[9] Unacceptably low ventilation rates were reported in more than half of the buildings investigated. ETS was associated with complaints in only six buildings, all of which also were affected by inadequate ventilation. Acceptable ventilation rates, according to the researchers, resulted in low levels of ETS constituents.

It is perhaps understandable, given the easy recognition of ETS, that persons experiencing sick building symptoms tend to

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blame ETS. Indeed, researchers have verified that the mere visibility or presence of tobacco smoke may provoke claims that ETS is the cause of reported symptoms and complaints.[10-11] However, removal of ETS through smoking bans may serve only to divert attention from more basic, underlying indoor air quality problems. As one commentator pointed out: "Removing the smoker entirely, then, may not affect health and comfort problems in 95% to 98% of sick buildings."[12]

Summary

The foregoing review illustrates the inconsistent and inconclusive nature of the data regarding claims about ETS exposure and disease in nonsmokers. Many of the reports relied upon by those who claim that ETS exposure is associated with disease in nonsmokers are epidemiologic studies that report relative risks which are deemed "weak" and rarely achieve statistical significance. The data in such studies are consistent with the hypothesis that there is no association between ETS exposure and disease. Many of those same studies invariably fail to consider other variables associated with disease, e.g., heredity, (poor) diet, occupational exposures, cross-infections, exposures to indoor and outdoor air pollution, etc. Moreover, nonsmoker exposures to ETS have not been directly assessed in any of the epidemiologic reports. Instead, kinds and amounts of exposure are assessed via

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questionnaire and depend upon recall by cases, next-of-kin and friends. This method is inherently crude and may lead to inaccuracies in exposure assessment. However, a large body of data from actual ETS measurements in the indoor environment exists. These data are examined in detail in the following section.

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III. ENVIRONMENTAL TOBACCO SMOKE AND INDOOR AIR QUALITY

It is often suggested that ETS is a major source of indoor pollution. However, the scientific literature indicates that, except under experimental or other extraordinary conditions, ETS does not have a significant influence on the quality of indoor air.[1-3] The claim that ETS is a significant source of indoor air pollution (and, as such, poses an increased risk for disease among nonsmokers) is based upon several unwarranted assumptions and misrepresentations of the scientific data. This becomes apparent in an analysis of the ETS/indoor air quality question, which addresses the nature and makeup of ETS, its contribution to indoor air, and the methods employed to measure exposure to it.

Sidestream Smoke

It is frequently claimed that sidestream smoke (SS), or the smoke from the burning end of the cigarette, contains much higher amounts of certain constituents than mainstream smoke (MS), or the smoke to which the smoker is exposed. Such a claim implies that a nonsmoker is at increased risk of disease because of his exposure to sidestream smoke, which has more allegedly "toxic" or harmful constituents than mainstream smoke. However, this argument is extremely misleading for several reasons, most notably because it fails to take into account that sidestream smoke is immediately

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diluted in the surrounding air. This diluted sidestream and exhaled mainstream smoke is more accurately called "ETS."

ETS differs chemically and physically from both mainstream and sidestream smoke.[4-9] ETS is a dynamic, ever-changing mixture which undergoes chemical reactions and physical changes as it ages and dissipates. Decay or disintegration patterns for ETS constituents vary over time and are dependent upon environmental features including air currents and attraction to surfaces, such as walls. In addition, reports indicate that constituents in ETS are hundreds to thousands of times more diluted than those identified in either SS or MS.[10-11] In fact, concentrations of ETS constituents in indoor settings frequently fall below detection limits of current scientific measurement devices.[12-16]

Many studies and reviews on ETS employ sidestream/mainstream smoke comparisons, ostensibly to demonstrate the kind and quantity of ETS constituents to which the nonsmoker is exposed. However, such comparisons are deceptive and misleading, as researchers on tobacco smoke chemistry reported in 1990:

> Although ETS originates from sidestream and exhaled mainstream smoke, the great dilution and other changes which these smoke streams undergo as they form ETS make their properties significantly different from those of ETS. Thus, the sidestream/mainstream ratios quoted . . . can be misleading if used out of context. The important question is not the

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> ratio of sidestream/mainstream but rather what is the concentration of the constituent in the indoor environment and how does it compare to levels from sources other than ETS. Studies based solely on observations of fresh sidestream, or highly and unrealistically concentrated ETS, should take into account the possible differences between these smokes and ETS found in real-life situations.[4]

Thus, it is both inappropriate and misleading to suggest that sidestream smoke is equivalent to ETS and that, under ordinary conditions, the nonsmoker is exposed to the levels of constituents reported in sidestream smoke.

Biological Markers

Some reports have suggested that the potential toxicity of ETS can be assessed by measuring the body fluids of nonsmokers exposed to ETS for mutagens, which are substances capable of altering the genetic structure of cells.[17-19] It is suggested that the presence of mutagens in body fluids, such as urine, may indicate that an individual has been exposed to substances capable of inducing cancer. These studies, however, did not employ realistic levels of exposure to ETS. Nor did they control adequately for the presence of mutagenic substances in the diet of the study subjects. Studies which have compared mutagens in the body fluids of nonsmokers exposed to realistic levels of ETS and nonsmokers not exposed to ETS report no significant difference in

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mutagenic activity.[20-23]

It is argued that sidestream smoke (and by inference, ETS) contains polycyclic aromatic hydrocarbons (PAHs), substances which have been designated as carcinogens or cancer-causing by various governmental agencies. However, in a series of papers, German researchers reported finding no significant differences in urinary PAH by-products between nonsmokers exposed to ETS and those not exposed.[24-26] Diet, on the other hand, was reported to have a profound influence on PAH by-product formation in all the study subjects.

It has also been suggested that DNA adducts can be utilized as biomarkers to assess exposure to ETS. (An adduct is a product derived from reactions between chemicals and biological material, such as DNA, the genetic material in the body). Research, however, does not support this theory; nonsmokers exposed to ETS do not appear to exhibit increased DNA adduct production.[27] Other studies report no increased chromosomal changes in the body fluids of nonsmokers exposed to ETS.[28-29]

Toxicity (Sidestream Smoke)

Animal inhalation experiments using fresh sidestream smoke or constituents of sidestream smoke fail to support the claim

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that ETS is a pulmonary carcinogen. Two such experiments have been published; both reportedly found no meaningful differences in the lung tissue of animals exposed to sidestream smoke and those not exposed to the smoke. In one of the studies, German scientists exposed rats and hamsters to very high levels of sidestream smoke for a 90-day period; they reported no significant physiological effects on the tissues of the animals.[30] In the second study, researchers from the American Health Foundation exposed hamsters to sidestream and mainstream smoke for 18 months; they observed no significant increase in lung tumors among the animals exposed to sidestream smoke.[31-33]

In addition, recent reviews of the literature on suspected pulmonary carcinogens have concluded that none of the individual constituents in sidestream smoke which are classified as potentially carcinogenic have been found to induce pulmonary cancer via inhalation in experimental animals.[34-35]

Constituents of ETS

A number of constituents typically have been cited in the literature to demonstrate the contribution of ETS to the indoor air. These include carbon monoxide, particulates, nicotine, and nitrosamines. However, the use of one or any combination of such constituents to determine ETS levels presents many problems. For

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example, although analytic and sampling methods continue to improve, there is at present no completely satisfactory and uniform procedure for measuring ETS.[36-37] Moreover, although the reported findings of studies which measure constituents in experimental conditions (e.g., in unventilated smog chambers) are frequently cited to dramatize the potential effect of ETS on indoor air, they have little, if any, similarity to those of studies which measure ETS in realistic settings. And finally, with the exception of nicotine, none of the constituents which have been used as substitute measures for ETS are characteristic of ETS alone. Other sources, such as heaters, stoves and furnishings, generate greater levels of those substances than those found in ETS.[38-40]

Exposure to ETS

Published studies indicate that nonsmoker exposure to ETS constituents under normal, everyday conditions is minimal. For example, researchers report that there is little difference in ambient levels of carbon monoxide (CO) in smoking and nonsmoking areas of workplaces and other public places and in homes with and without smokers.[12,15,38,41-44] Other studies indicate that ETS contributes less than half of the total particles in the air of an average public place.[2,13-15,45-49] Typical measurements of nicotine, which is used as a marker for ETS exposures because it is unique to tobacco smoke, range from an exposure equivalent of one

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one-hundredth (1/100) to less than one one-thousandth (1/1000) of a filter cigarette per hour.[13-16,50-56] This means that a nonsmoker would have to spend from 100 to 1,000 hours in an office, restaurant or other public place where smoking is permitted in order to be exposed to the nicotine equivalent of a single cigarette.

Carbon Monoxide

Research has shown that the main sources of CO in the outdoor urban environment are motor vehicles and industrial processes,[57] and that indoor levels are affected by these outdoor sources, mainly through ventilation, and by numerous activities such as cooking and heating. In fact, studies have indicated that gas stoves in kitchens and heating units are often major sources of CO in homes.[38,58-59]

After their review of such studies, participants at the University of Geneva symposium on ETS in 1983 concluded that CO from environmental tobacco smoke "is not important from a health point of view."[60] Similarly, researchers from the Lawrence Berkeley Laboratory in the U.S., who are otherwise critical of tobacco smoke, concluded that "based on theoretical and empirical results, CO sidestream emissions from cigarettes have often been overemphasized."[42]

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Particulates

A paper published in a 1980 issue of Science magazine, in which the authors reported the results of their efforts to measure particles or particulates in the air of smoking and nonsmoking areas, is often cited to support the claim that ETS is a major indoor pollutant.[61] The authors, Repace and Lowrey, contend that the levels of particles they observed in smoking areas were much higher than in the nonsmoking areas. However, their study results are inconsistent with many others. For example, the average particle count attributed to ETS in their study was from three to more than 20 times higher than the average levels reported in other studies of office buildings, restaurants and residences.[2,13-15,48,62-64]

There are a number of explanations for Repace/Lowrey's apparent overestimation of ETS exposure. First, they selectively sampled environments such as meeting and game rooms, bars and sandwich shops which do not represent normal occupancy conditions and where particulate levels are likely be high regardless of the presence or absence of tobacco smoke. Second, through inappropriate testing methods, they incorrectly assumed that all particles in the air arose from ETS. However, as several researchers have noted, ETS typically contributes less than half of the overall particle levels reported in indoor spaces,[45-47]

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perhaps because particles are also generated by people and their everyday routine activities such as movement and cooking.[65-66]

/In addition to ignoring potential indoor sources of particles, the study has been criticized on other grounds. For example, Repace and Lowrey did not measure ventilation rates or humidity, and they failed to clean and calibrate their testing instrument prior to making their measurements.[66] Indeed, the testing device they used "is no longer recommended" for measuring tobacco smoke.[36-37]

In their report, Repace and Lowrey implied that the particle levels attributed to ETS are potentially harmful to the health of nonsmokers. However, an earlier study by Bouhuys, et al., reportedly found "no evidence that high TSP (total suspended particulates) levels in homes with smokers were associated with increased symptoms or lung function loss among nonsmokers in the same home."[67] Binder, et al., also reported that respirable particulate levels in homes were "independent of the presence or absence of respiratory disease."[68] More recently, Lebowitz, et al., reported on data which showed that respiratory symptoms in asthmatics were associated with total indoor particle levels (e.g., dust), but not with the presence of tobacco smoke.[63,69-70]

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Particulates and Radon

A theory suggesting that concentrations of radon decay products increase in the presence of tobacco smoke, thus implying an increased risk of lung cancer for nonsmokers, has been reported in the literature.[71-73] The theory suggests that radon decay products attach to particles (including ETS) in the air, remain suspended, and are subsequently taken up in the lungs of nonsmokers. However, data from actual research studies indicate that it is the unattached, gaseous fraction of radon which determines the amount of radiation to which the respiratory tract is exposed.[74-76] According to these data, the unattached fraction of radon daughters will decrease as dust or particulate levels increase, thereby lowering the potential dose of radiation to the lungs.

Nicotine

Since ambient nicotine is produced almost exclusively by burning tobacco, it is considered to be a more reliable indicator of the amount of tobacco smoke in the environment than other smoke constituents. Studies which have used nicotine in this way suggest that tobacco smoke contributes very little to the indoor atmosphere.[12-16,50-56] For example, Drs. William Hinds and Melvin First of the Harvard School of Public Health reportedly found very small

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amounts of nicotine in the atmospheres of bars, bus and airline terminals, restaurants, and cocktail and student lounges.[50] French researchers, using a different method of measuring nicotine to assess the amount of tobacco smoke in the atmosphere, reported finding higher concentrations of nicotine than Hinds and First.[51] However, they still concluded that "smoking does not present a risk to nonsmokers."

Other studies have reached similar conclusions. In 1984, Japanese researchers tested a personal nicotine monitor in a number of public places, including offices, restaurants, lobbies, terminals and public transportation. They reported levels of nicotine exposure equivalent to one one-thousandth (1/1000) to four one-hundredths (4/100) of a cigarette per hour.[53] Their findings were repeated in 1987.[54]

A year later, scientists from IT Corporation, a firm specializing in the assessment and reduction of environmental substances, measured nicotine in offices and restaurants in Ottawa, Canada.[13] They reported average nicotine exposure levels equivalent to three one-hundredths (3/100) of a cigarette per eight-hour workday, and three one-thousandths (3/1000) of a cigarette during a one-hour meal.

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In a nationwide sampling survey in the United Kingdom, researchers monitored nearly 3,000 sites in travel, work, home and leisure locations for ambient nicotine, CO and particle levels.[12] Smoking was known to have occurred at almost half of those sites, yet in three-fourths of the samples, nicotine levels were too low to be detected. Canadian researchers also reported levels of nicotine at or below levels of detection even in locations with recirculated air from designated smoking areas.[64]

There are few data that suggest that the tiny amounts of nicotine to which a nonsmoker may be exposed are related to human disease. For example, two German scientists monitored several physiological responses in nonsmokers exposed to tobacco smoke under laboratory conditions.[78] They concluded that the amount of nicotine to which their subjects were exposed was too small to alter sensitive test measurements of heart rate, heart muscle tracings (EKG), blood pressure or skin temperature. More recent research tends to support these conclusions. In a 1983 study, researchers who measured exposures of nonsmoking flight attendants to nicotine during transpacific flights concluded that the concentrations were so small that they were "unlikely to have physiologic effects."[56] In 1986, researchers monitored levels of nicotine, CO and particulates in 66 commercial flights in the U.S. They concluded that the non-smoking sections of commercial aircraft were essentially free of ETS.[16]

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Other Constituents

Studies which have examined levels of nitrosamines, nitrogen oxides and volatile organic compounds (such as benzene) in ETS report that it makes minimal contributions to overall ambient air levels in homes, the workplace and public places.[38-40,48,79-88] However, this has not prevented claims from being made that such exposures are harmful to the nonsmoker.

For example, it has been claimed, largely on the basis of reports by Brunnemann, et al., that nitrosamines from tobacco smoke represent a cancer risk to nonsmokers.[89-90] It has also been claimed that nitrosamine levels in sidestream smoke exceed those found in mainstream smoke, with the implication that those levels are also found in ETS. However, this is not the case. In 1982, Austrian scientists reported that their measurements of nitrosamine levels in ordinary public places were far below the values reported earlier by Brunnemann and his co-workers. They emphasized that the levels of nitrosamines which may occur in environmental tobacco smoke "are quite different" from those found in sidestream smoke.[80]

One scientist who reviewed the literature stated that nitrosamines occur everywhere "independent of any type of tobacco

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combustion, in which case concentrations are often reached in respiratory air that are far above those that may be produced by tobacco smoke."[91] He concluded that, as a consequence, any claimed implications of health risks from nitrosamines in tobacco smoke "clearly move into the range of speculation." Another scientist, Dr. Helmut Schievelbein, Director of the Institute of Clinical Chemistry in Munich, Germany, observed that concentrations of nitrosamines either consumed or generated daily by individuals are "many times greater" than those found in environmental tobacco smoke.[79]

Other constituents which have been identified in analyses of sidestream smoke are often assumed to contribute substantially to indoor air in the form of ETS. The most commonly mentioned constituents include nitrogen dioxide (NO_2), formaldehyde and volatile organic compounds. However, these compounds are present in very small amounts in ETS.[39-40,48] Recent studies report no correlations between ETS and levels of NO_2 and volatile organic compounds in residences and offices.[39,48,83-84] Indeed, research indicates that levels of these components generated from other ordinary sources (e.g., cooking stoves, heaters and building materials) are much greater than those contributed by cigarette smoking.[39,83-84]

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Reports have also suggested that exposure to benzene from ETS is substantial.[92-93] However, a recent study by German investigators reported that benzene uptake by nonsmokers exposed to ETS is negligible compared to background levels from other environmental sources.[85] Other scientists, who compared benzene emissions from cigarettes with those reported from automobiles, have observed that "an average person is potentially exposed to 1,000 times more ambient benzene from one automobile than from a smoker in a given year."[94]

Cotinine as a Marker for ETS Exposures

It has been reported that cotinine, a substance metabolized or converted from nicotine by the body, can be used as a biological marker to measure nonsmoker exposure to ETS.[95-96] While some reports suggest that cotinine is a reliable marker for total exposure to tobacco smoke, many others do not for a variety of reasons.[97-106] For example, it has been reported that individuals metabolize nicotine in different ways at different times and that elimination rates for cotinine vary among individuals. In addition, recent research indicates that diet may contribute to levels of nicotine and cotinine found in the body, thereby interfering with reported exposure levels from nicotine in the ambient air.[107] Scientists have also noted that different methods of analysis may influence final recorded levels of

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cotinine.[108] And finally, it has been observed that because nicotine is largely present in the gas-phase of ETS, measurement levels of its metabolite, cotinine, do not reflect exposures to other constituents present, for example, in the particulate phase of ETS.[99]

For these reasons, cotinine should not be regarded as a reliable quantitative measure of ETS exposure. At best, cotinine may be used as a qualitative marker of ambient nicotine exposures.

Other Determinations of ETS Exposure/Thresholds

Despite the difficulties researchers have encountered in determining ETS exposure, it has been suggested that the nonsmoker's risk of disease attributable to ETS can be estimated by comparing the levels of exposure to ETS with the active smoker's exposure to mainstream smoke. These estimates are derived by extrapolating the exposure (and, thus, the imputed risks) of active smoking to nonsmoker exposure to ETS. However, such an extrapolation is based upon several unwarranted assumptions.[109] First, it assumes that mainstream smoke and ETS are chemically and physically comparable. The foregoing discussion demonstrates that this is not the case. Indeed, even the 1986 Report of the Surgeon General on ETS conceded:

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> Comparison of the relative concentrations of various components of SS and MS smoke provides limited insights concerning the toxicological potential of ETS in comparison with active smoking. . . . SS characteristics, as measured in a chamber, do not represent those of ETS, as inhaled by the nonsmoker under nonexperimental conditions.[110]

Similarly, the 1986 NAS Report on ETS concluded:

> Because the physicochemical nature of ETS, MS, and SS differ, the extrapolation of health effects from studies of MS or of active smokers to nonsmokers exposed to ETS may not be appropriate . . . .[111]

Second, it assumes the validity of a "linear extrapolation" from active smoking to nonsmoker exposure to ETS. That is, it assumes that the level of exposure to active smoking (and the implied risk) for the smoker can be utilized to calculate the implied risk of the nonsmoker on the basis of his proportionate exposure to ETS. This model, however, has never been verified experimentally, nor has it been demonstrated to be applicable to ETS or its constituents.[112]

In fact, dose-extrapolation models which are based upon realistic estimates of exposure and dose for nonsmokers fail to predict an appreciable increased risk of disease for nonsmokers exposed to ETS.[113-114,116] It has been estimated, for example, that a nonsmoker is exposed to approximately two-hundredths of one

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percent (0.02%) of the particulates to which an active smoker is exposed.[11,109,113-115] Consequently, the reported risk estimates are typically many times lower than risk estimates which are based upon the epidemiologic or population-based studies of ETS. These two different methods of risk assessment lead to markedly incompatible results. Indeed, the Project Officer for the EPA's draft risk assessment on ETS revealed that the drafters of the report were "unsuccessful" in their attempts to estimate a dose-response relationship by extrapolating from active smoking (mainstream smoke) to ETS exposure, and that, as a result, they decided to "present other methods" to estimate the risk of disease from ETS exposure.[117]

Summary

A review of the published literature on ETS demonstrates that exposure to ETS in typical indoor environments is minimal. Yet tobacco smoke, because it is visible, has become a target for those who apparently want a quick, simple solution to the problem of indoor air pollution. However, as one Canadian scientist observed, "the claim that smoking is responsible for indoor air pollution is an oversimplification of a complex, multi-source problem."[66]

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IV. ENVIRONMENTAL TOBACCO SMOKE AND THE PUBLIC

> There is a new segment emerging in our society -- that of the social engineer, bureaucratic wowser, or health fanatic. A group of people who are attempting to enforce their beliefs on a community which has the right to choose what to eat, drink and smoke.[1]

Exposure to environmental tobacco smoke under extreme conditions may provoke complaints of irritation and annoyance. However, the mere sight and smell of tobacco smoke may be enough to produce strong reactions in individuals who intensely dislike smoking. Although the extent to which environmental tobacco smoke may bother or annoy individuals under normal conditions has not been established scientifically, it has been suggested that extreme reactions to tobacco smoke exposure may have a strong emotional or psychological basis.[2-4] Whatever the reasons for such reactions, the literature on this subject suggests it is misleading to claim, as antismokers frequently do, that such subjective or emotional reactions have a physical basis.

Governmental Regulations and Individual Rights

Although antismoking organizations claim to seek protection for the health and welfare of the nonsmoker, their efforts to make smoking socially unacceptable are part of a broader objective -- the severe restriction or even prohibition of smoking by

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governmental legislation. Of course, such regulations restrict the freedom of the smoker, and antismokers are compelled to justify this kind of political control of individual behavior. They have accomplished this, in large measure, by exploitation of the so-called "health" argument involving ETS exposures to nonsmokers.

For example, the 1986 U.S. Surgeon General's Report contends that ETS causes lung cancer in adult nonsmokers and respiratory conditions in children, and that the separation of smokers and nonsmokers is not an effective method of minimizing the nonsmoker's exposure to ETS.[5] The Report concludes that smoking bans will not only reduce ETS exposures, but will also "alter smoking behavior and public attitudes about tobacco use." The Report further suggests that "over time, this may contribute to a reduction of smoking." Thus, the underlying motivation for the use of the ETS/health argument is to attain a "smoke-free society by the year 2000."

The Surgeon General's tactics, however, have been questioned by a number of critics. One reviewer suggested that the Surgeon General's conclusions were based on "flimsy" evidence presented in an effort to "divert attention" from important health concerns such as the "poisoning of the environment."[6] A U.S. Congressman, in a letter to the Congressional Record, wrote that "the conclusions in the Surgeon General's Report are not supported

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by the research in his own report."[7]

Moreover, the Surgeon General's claim that separation of smokers and nonsmokers does not minimize nonsmoker exposure to ETS is without scientific support. Studies aboard commercial aircraft and in offices indicate, contrary to the Surgeon General's Report, that the simple separation of smokers and nonsmokers effectively minimizes nonsmoker exposure to ETS.[8-16] One recent study, for example, reported that the use of designated smoking areas reduced exposure to ETS by 95 percent.[8] Another study of a smoking-restricted office building reported that ambient nicotine in nonsmoking areas was virtually undetectable, suggesting that ETS had a negligible impact on the nonsmoking areas in the building.[9] In addition, Canadian researchers, in a series of studies, collected data on levels of ETS constituents in offices with different smoking regulations. They reported no significant differences in average ETS constituent levels between nonsmoking offices that received recirculated air from designated smoking areas and nonsmoking offices that did not receive recirculated air.[10-11,14,16] They concluded:

> The results indicate that the provision of a designated, but not separately ventilated smoking area can effectively eliminate or drastically reduce most components of environmental tobacco smoke from nonsmoking offices.

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Similarly, restrictions and bans on smoking aboard commercial aircraft have been imposed despite the availability of data suggesting that the simple separation of smokers and nonsmokers effectively reduces exposure to ETS. A case in point was the 1989 decision by the United States Congress to ban smoking aboard flights of six hours or less. This Congressional action was taken before the completion of a large study of inflight exposures to ETS commissioned by the U.S. Department of Transportation (DOT) and without any evidentiary or other hearings. When the study was released several months later, it contained data indicating that individuals seated in nonsmoking sections are exposed to extremely low levels of ETS constituents.[17] In fact, many measured levels of ETS constituents were reported to be below the detection limits of the air sampling monitors. In a recent presentation, one of the principal scientists responsible for the study questioned "whether or not Congress knew that the ETS results were not strongly compelling prior to the study's release and as a result preempted the use of the results in the deliberations on whether or not to make the ban permanent."[18]

Research results and technical reviews available prior to the Congressional action also suggest that it was not based upon scientific data.[12,15,19-20] In 1988, a review of the relevant data concluded that "the available scientific evidence does not support the prohibition of smoking on commercial aircraft."[19] The review

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stated that the data "suggest that factors or substances other than ETS may be major contributors to subjective complaints of discomfort by passengers and flight crew." Similarly, in 1989, an Australian writer concluded that the available data "do not lend support to the hypothesis that exposure to environmental tobacco smoke may present a risk to the health of cabin staff or passengers."[20] He further observed that providing smoking and nonsmoking sections "meets the reasonable requirements of passengers."

One of the most comprehensive assessments of aircraft cabin air quality to date was undertaken in Europe in 1989.[12] The results indicate that total exposure to ETS aboard aircraft is "rather small and insignificant in comparison to total life exposure to air pollution." The researchers conducting the study concluded that any possible health effects from ETS exposures were "not likely to have been elicited" by exposures aboard flights. They also noted that reports of irritation and annoyance commonly attributed to ETS exposures may have been "potentiated by the low humidity, high temperature and high carbon dioxide levels found."

Governmental Reports

Governmental reports throughout the world, like the 1986 Report of the Surgeon General, have done little to clarify the

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scientific nature of the ETS issue for the public. In the United Kingdom, for instance, the Fourth Report of the Independent Scientific Committee on Smoking and Health (1988) endorsed the heavily criticized claim that ETS increases the nonsmoker's risk of lung cancer from 10 to 30 percent compared with the non-exposed nonsmoker,[21] although, as one commentator noted, "exposure data are very unreliable, and there is a proven basis for bias in the direction of exaggeration of the risk."[22] Still another U.S. governmental report concluded that exposure to ETS increases the risk of disease among nonsmokers, even though, when the authors sought to identify both the valid data and the areas of uncertainty regarding ETS, they observed "many more of the latter . . . than the former."[23] In addition, the most recent monograph on tobacco smoke from the World Health Organization's International Agency for Research on Cancer (IARC) warns of increased risks of disease among nonsmokers exposed to ETS while conceding that the epidemiological data are consistent "either with an increase or with an absence of risk."[24]

In June 1990, the U.S. Environmental Protection Agency (EPA) issued a public review draft risk assessment for ETS.[25] The review draft concluded that ETS is a cause of lung cancer in adult nonsmokers and respiratory disease in children, and that ETS is a Group A ("known human") carcinogen. The draft also estimated that ETS exposures are responsible for 3,800 lung cancer deaths a

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year among nonsmokers in that country. The draft was heavily criticized during the public review period by numerous scientists and researchers in the area, most notably because its authors failed to follow the EPA's own guidelines for risk assessment, in addition to addressing selectively the scientific literature and drawing conclusions which were not supported by the scientific data.[26] Despite the overwhelming scientific criticism, the Science Advisory Board, a review panel for the EPA, agreed with the conclusions in the draft and submitted its endorsement to the administrator of the agency. The director of the EPA will decide whether or not the draft will become part of the agency's policy.

In July 1991, the U.S. National Institute for Occupational Safety and Health (NIOSH) issued a "Current Intelligence Bulletin" on ETS in the workplace.[27] The Bulletin concludes that ETS is a potential occupational carcinogen and recommends that exposures be reduced "to the lowest feasible concentration." However, the Bulletin did not examine the data on actual workplace exposures to ETS or the information on workplace exposures to ETS which can be found in 11 studies on ETS exposures and lung cancer in nonsmokers. Those data provide no support for NIOSH's claims.

In June of 1991, the Environment Committee of Great Britain's House of Commons issued a report, entitled Indoor Pollution, which contained recommendations for government action

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on improving the indoor environment.[28] Prior to issuing its report, the committee heard presentations on ETS from individuals and organizations with widely varying views on the issue, including scientists with the British-American Tobacco Company (BAT) and representatives of the antismoking group Action on Smoking and Health (ASH). In its report, the committee acknowledged that there is a "scientific dispute" about the purported health effects of ETS, but urged that efforts be made to minimize nonsmoker exposure to ETS, and that children, in particular, should be "protected."

Still another recent governmental report on ETS, issued to the Minister and State Secretary for Welfare, Health and Cultural Affairs in the Netherlands, presented a critical assessment of the scientific literature on ETS.[29] While concluding that exposure to ETS increases the risk of disease in nonsmokers, the report also concluded, contrary to the U.S. EPA draft risk assessment, that the various "distorting factors" in reports on ETS preclude the possibility of making a well-founded estimate of the magnitude of so-called increased risk from ETS exposure.

Court Decisions

However, some governmental bodies have refused to give in to the emotional rhetoric of antismoking organizations and public health officials. For example, courts in the United States have,

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with few exceptions, denied the claims of individuals who contend they have a "right" to a smoke-free workplace. A law journal article noted that "the efforts of nonsmokers to restrict workplace smoking have met with little success in the courts, which have been reluctant to supplant the roles of employer and employee."[30]

Courts in other countries have also ruled on related issues. In 1987, the Supreme Court of Canada refused an appeal by a worker who sought a smoke-free environment on the grounds that ETS is a "dangerous" workplace substance.[31] Similarly, in Japan, a court judgment denied a plaintiff's request that more than half of all the railway cars on passenger trains be designated nonsmoking because, the Court ruled, ETS exposures do not "exceed the limits of normal tolerance."[32]

In another recent U.S. court case, an antismoking organization challenged the decision of the U.S. Occupational Safety and Health Administration (OSHA) not to issue an emergency temporary standard banning tobacco smoking in the workplace. An Appeals Court upheld the OSHA decision, stating that the agency "reasonably determined that it could not at this time sufficiently quantify the degree of risk associated with workplace exposure to ambient tobacco smoke to justify issuing an emergency temporary standard."[33]

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In 1990, the District Court of the Hague in the Netherlands ruled that the government-funded Dutch Foundation on Smoking and Health (StiVoRo) was not liable to the tobacco industry for its statement that ETS is "unhealthy" for nonsmokers. The Court recognized, however, that:

> In the present discussion over the consequences of passive smoking, the starting point must be that scientific investigation has not succeeded in establishing which facts are true and which are not true. It is evident from the documents that the discussion concerns a controversial subject, on which there are divergent - whether or not scientifically supported - opinions. From the documentary evidence it is likewise apparent that the results of medical research and positions taken within medical science with respect to the consequences of passive smoking also vary.[34]

In 1985, the Supreme Insurance Court of Sweden awarded compensation for occupational injury to the estate of a nonsmoker who worked with smokers and who subsequently died of lung cancer. Although the media described the Court's decision as a causal indictment of ETS exposure, one commentator noted that the decision was based upon the structure of occupational injury regulations in Sweden and not upon any scientific evidence of a causal relationship between lung cancer and ETS exposures.[35] This author explained that the Swedish insurance system operates under a reverse burden-of-proof provision, namely, that the relationship between a suspected factor and an injury is assumed to be causal unless

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substantially stronger reasons are produced to suggest otherwise. Consequently, this provision does not require the claimant to present scientific proof of a causal relationship between a suspected factor and an injury to be awarded compensation.

Three years later, in Australia, a city bus driver who contended his lung cancer was caused, at least in part, by exposure to ETS on the job made a claim against the Melbourne Transit Authority.[36] The matter was settled by the insurer of the Transit Authority. This settlement was interpreted by some as establishing a legal precedent for the claim that ETS exposure has been proven to cause lung cancer. However, the bus driver's claim regarding ETS was only part of his complaint, and the tribunal before which the proceeding was held heard only part of the evidence in the case before the insurer made a settlement offer.[37] Thus, this settlement established no formal legal precedent regarding ETS health claims.[36-37]

Several other recent court decisions have been heavily touted by antismoking organizations as important, precedent-setting cases regarding the adverse health effects from exposure to ETS. However, a careful examination of the context in which each of these cases was heard reveals the limited nature of the claims. For example, in April 1990, a social security commissioner in the U.K. ruled that an asthmatic employee of the social security

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administration had suffered specific adverse reactions after exposure to ETS.[38] However, the Commissioner's ruling pertained only to the question of whether or not an "accident" had occurred under the meaning of the 1975 Social Security Act.[39] The decision made no finding regarding the medical facts of the case, and no finding as to whether the claimant is disabled or whether any benefits will be granted. Indeed, the Commissioner noted that his decision establishes "no precedent for other cases" involving claimed ETS exposures. In addition, he noted that this was an "unusual case" with no application to the "ordinary case of alleged injury or disease" from exposure to ETS.[39]

Most recently, a court in Australia ruled in favor of the Australian Federation of Consumer Organizations (AFCO) in its allegation that a July 1986 advertisement on ETS by the Tobacco Institute of Australia (TIA) was false and "misleading and deceptive" under that country's Trade Practices Act. The case centered around the TIA's statement in the ad that "there is little evidence and nothing which proves scientifically that cigarette smoke causes disease in nonsmokers."[40] The Tobacco Institute of Australia has appealed the decision on the basis that their statement is "an opinion which has a rational scientific basis."[41]

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Summary

Governmental involvement in the ETS question raises many disturbing questions. An Australian writer who recently addressed the issue cautioned that:

> Those involved in government anti-smoking activities should be aware of the tenuous nature of the data on passive smoking and health effects. The deliberate use of fear inducing tactics by misrepresenting such data by dubious extrapolations is propaganda and not health education.[42]

Similarly, a group of physicians in the U.S. observed that "scientific data have not always been utilized objectively by governmental agencies or regulatory bodies that have their own inherent public health or political agenda."[43] They concluded:

> The implementation of public policies must be based on good science, to the degree that it is available, and not on emotion or on political needs. Those who develop such policies must not stray from sound scientific investigations, based only on accepted scientific methodologies. Such has not always been the case with environmental tobacco smoke.

It is clear that once claims regarding environmental tobacco smoke and disease in nonsmokers are placed in their proper scientific perspective, the issue of prohibiting smoking in public places becomes a social and political one. The issue thus involves

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An alternative to governmental regulation of smoking supports the notion of proper ventilation for all building occupants together with the promotion of courtesy and cooperation among smokers and nonsmokers. This solution is supported by data on indoor air quality and relies upon common sense, courtesy and the free marketplace for decisions concerning smoking. More importantly, however, this solution avoids unnecessary governmental interference with its subsequent loss of individual freedom.

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whether or not public policy should mandate the prohibition of smoking because it is seen by some as a nuisance or annoyance. If the decision is reached in favor of regulation, then one must decide whether the same line of reasoning applies to other kinds of "annoyances" encountered in everyday life and whether those "annoyances" should also be banned. The implication of such reasoning should be obvious to anyone who is opposed to unwarranted governmental intrusion into people's lives. As a German scientist recently concluded:

> Whether a real risk is involved which in the future can be demonstrated on the basis of measurable results remains an open question. In light of this health officials should concentrate on more significant environmental problems in the long term interest of society rather than wasting time and money on trivial issues.[44]

Conclusion

Smoking bans do not improve fresh air ventilation, nor do they ensure acceptable indoor air quality. Complaints about the quality of indoor air typically arise in poorly ventilated areas. For example, data from over a thousand "sick building" investigations in North America and Europe indicate that more than half of the buildings were inadequately ventilated, and that ETS was associated with occupant complaints in no more than four percent

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of all the cases. (For a discussion of this data, see the section on "Sick Building Syndrome.")

The importance of adequate ventilation for maintaining indoor air quality has been recognized by many researchers and organizations. The experience of the American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE), during its attempt to establish a ventilation standard which balances the need for adequate ventilation with the demand for little or no exposure to ETS, is instructive in this regard. Initially, the organization developed a standard which established ventilation rates solely on the basis of the presence or absence of smokers. However, the prescribed ventilation rates for nonsmoking areas were reported to be inadequate. Consequently, in 1989, the organization published a ventilation standard entitled "Ventilation for Acceptable Indoor Air Quality" (ASHRAE Standard 62-1989) which adopts a single ventilation rate with no distinction between smoking and nonsmoking areas.[45] According to knowledgeable researchers, the standard serves as a guide for preventing indoor air problems and for establishing ventilation rates for indoor areas in order to "control carbon dioxide and other contaminants with an adequate margin of safety, and to account for variations among people, varied activity levels, and a moderate amount of smoking."[46-48]

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